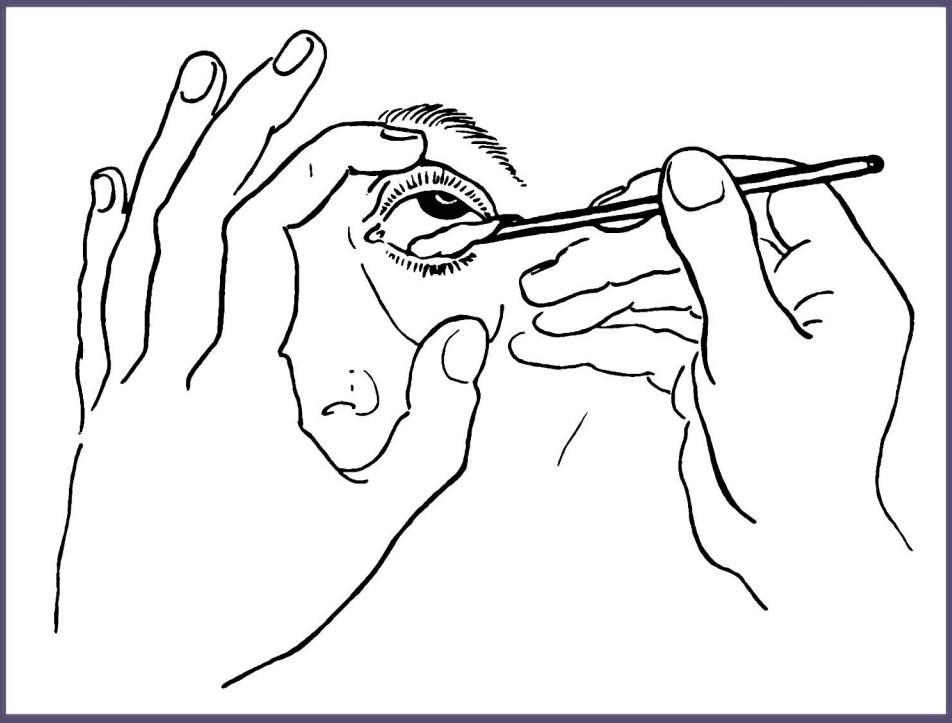
M. ZOLOTARYOVA

DISEASES of the EYE



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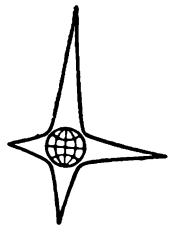

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ГЛАЗНЫЕ БОЛЕЗНИ

ИЗДАТЕЛЬСТВО „МЕДИЦИНА“ МОСКВА

На английском языке

M. ZOLOTARYOVA

DISEASES of the EYE

Translated from the Russian

by

YURI SHIROKOV

MIR PUBLISHERS • MOSCOW • 1966

UDC 617.7 (075.3) = 20

CONTENTS

Introduction 9

1. OUTLINE OF THE ANATOMY OF THE ORGAN OF VISION 11

Protective Structures and Appendages of the Eye 11
The Eyeball 17
Muscles of the Eye 24

2. THE VISUAL FUNCTIONS AND METHODS OF EXAMINATION 26

Central Vision and Visual Acuity 26
Colour Vision 30
Peripheral Vision and Field of Vision 32
Photoreception 35
Binocular Vision 35

3. REFRACTION AND ACCOMMODATION 37

Ocular Refraction. Optic Lenses 37
Accommodation 39
Clinical Picture of Refraction Anomalies. Selection of Spectacles 41

4. LAYOUT AND EQUIPMENT OF THE OPHTHALMOLOGIST'S OFFICE OF AN OUT-PATIENT CENTRE AND THE EYE DEPARTMENT OF A HOSPITAL 46

The Ophthalmologist's Office 46
Layout and Equipment of the Eye Hospital 49
Preoperative Treatment. Surgery and Postoperative Care . . 53

5. EXAMINATION OF THE PATIENT 56

History Taking 56
Examination of the Eyelids, Conjunctiva, Cornea, and Iris 57
Ophthalmoscopy 63
Determination of the Intra-Ocular Tension 65

6. METHODS OF TREATING EYE DISEASES 68

General Therapy 68
Physiotherapy, X-ray and Radiotherapy 72
Local Therapy 72
Astringent and Caustic Drugs 76
Antiseptic Drugs 76
Resolvents and Irritants 77
Anaesthetics 78
Mydriatics and Miotics 78
Corticosteroids 79
Dressings 80

7. DISEASES OF THE EYELIDS AND LACRIMAL APPARATUS 82

Diseases of the Eyelids 82
Diseases of the Skin of the Eyelids 82
Diseases of the Margins of the Eyelids 83
Changes in the Position and Movements of the Eyelids 85
Diseases of the Lacrimal Apparatus 88
The Lacrimal Passages and Their Diseases 89
Inflammation of the Lacrimal Sac 91

8. DISEASES OF THE CONJUNCTIVA 95

Conjunctivites of Exogenous Origin 96
A. Acute Conjunctivites 96
B. Inflammation of the Conjunctiva Due to Mechanical, Chemical and Physical Factors. 102
C. Conjunctivites of Endogenous Origin 102
Degenerative Changes of the Conjunctiva 104

9. TRACHOMA 105

The Clinical Picture and Diagnosis of Trachoma. . . . 105
Differential Diagnosis Between Trachoma, Folliculosis and Follicular Conjunctivitis 108
The Aetiology and Prevalence of Trachoma 111
Organization of Trachoma Control 112
Treatment of Trachoma 113

10. DISEASES OF THE CORNEA AND SCLERA 116

Diseases of the Cornea 116
Superficial Keratitis 118
Scrofulous Keratitis 119
Neurogenous or Neurotrophic Keratites 121
Herpetic Keratitis 121
Epidemic Keratoconjunctivitis of Adenovirous Aetiology 122
Creeping Ulcer of the Cornea (Ulcus Serpens) 122
Deep or Interstitial Keratites 125
Deep Tuberculous Keratitis 126
Keratites of Malarial and Brucellar Aetiology 127
Diseases of the Sclera 128

11. DISEASES OF THE UVEAL TRACT 130

Iritis 130
Iridocyclitis 131
Choroiditis 133
Endophthalmitis 133
Newgrowths of the Uveal Tract 134

12. DISEASES OF THE CRYSTALLINE LENS AND THE VITREOUS BODY 135

Diseases of the Crystalline Lens 135
Cataract 135
Diseases of the Vitreous Body 139

13. GLAUCOMA 140

The Clinical Picture and Classification of Glaucoma . . . 141
Acute Attack of Glaucoma 143
Diagnosis, Prophylaxis and Treatment of Glaucoma . . . 145

14. DISEASES OF THE RETINA AND OPTIC NERVE . . . 149

Diseases of the Retina 149
Circulatory Disorders in the Retina 149
Pathological Changes of the Retina and Retinal Vessels in Certain Systemic Diseases 150
Pigmentary Degeneration of the Retina 151
Detachment of the Retina 151
Newgrowths of the Retina 152
Diseases of the Optic Nerve 152

15. DISEASES OF THE EYE MUSCLES 155

Diseases of the Orbit 155
1. Diseases of the Eye Muscles 155
Concomitant Strabismus 155
Paralytic Strabismus 156
Nystagmus 156
2. Diseases of the Orbit 156
Orbital Periostitis 157
Orbital Phlegmon 157
Orbital Tumours 157

16. EYE INJURIES 159

Mechanical Injuries. 160
Thermal and Chemical Burns 164
Prevention of Eye Injuries 166
Prescriptions 169
Index 173

INTRODUCTION

Ophthalmology is a branch of medicine concerned with diseases of the eye and the anatomy and physiology of the organ of vision—the optic analysor, its optic properties and functions. The optic analysor is the collective name given to the various parts of the organ of vision: the image-producing part known as the retina, the optic nerve which beyond the optic chiasm in the brain is continuous with the optic tract, and the subcortical and cortical centres in the brain.

In the mid-19th century, ophthalmology became an independent branch of medical science; before this diseases of the eye had been dealt with by surgeons. This separation of ophthalmology was necessary in view of the highly important role vision plays in the life of man, the structural complexity of the optic analysor, the variety of its functions and other specific features, which require special methods of examination and therapy. Vision is extremely important to man because without it he cannot carry out useful work, read and write, or have everyday relations with other people, etc. The organ of vision is often involved in diseases of systems and individual organs, metabolic disorders, etc.

Pathological changes in the organ of vision may signal the presence or imminence of certain diseases.

The origin, course and spread of eye diseases depend on the way of life and social factors, occupational hazards, and the general standards and hygienic habits of the population. According to S. S. Golovin (1910), there were about 300,000 blind people in prerevolutionary Russia. The causes of blindness were trachoma, gonoblennorrhea, smallpox, syphilis, and other diseases. After the Great October Socialist Revolution, the fundamental change in the country's economy was accompanied by a tremendous advance in the general and sanitary conditions of the population, and in the standards of medical services, specialized medical aid in particular.

The emphasis on prophylaxis in Soviet medicine, and the measures taken for early detection and medical treatment of cases have resulted in a sharp decline in the incidence of many diseases, especially infectious ones, including those of the eye. For example, blindness due to smallpox, diphtheria, gonoblennorrhea, measles, and other infections is no longer encountered in the USSR, while trachoma has been nearly wiped out. Loss of vision due to trachoma, which was the largest single cause of blindness in tzarist Russia is today found almost exclusively among old people who developed this disease in childhood and were denied adequate treatment.

The introduction of standard rules for protection and safety has reduced the hazards of eye injuries in industry. The continuous progress of medical science, which is inseparably linked with practice, is playing a great part in the effective control of blindness in the USSR. The achievements of Soviet ophthalmology are largely due to the work of the prominent scientists: L. G. Bellyarminov, M. I. Averbach, V. P. Filatov, V. V. Chirkovsky, and V. P. Odintsov, to mention a few.

The development of Soviet ophthalmology, both theoretical and practical, has been greatly assisted by the network of ophthalmological research institutes and clinics, eye hospitals and hospital departments for eye diseases, clinico-prophylactic centres (dispensaries) and consulting-rooms set up under Soviet government.

Today ophthalmological treatment is available to all. For example, ophthalmological departments are found at any regional hospital, and consulting-rooms at any clinic. In a district, the ophthalmological service is directed by the district oculist, who attends to out-patients and inmates of eye hospitals, and directs the general health service in matters within his competence. In a number of regions where trachoma still lingers there are specialized ophthalmological stations run by a doctor's assistant or a medical nurse.

The role of the general health service in controlling eye diseases and blindness is very important, particularly in rural areas. Any medical worker should be capable of assisting an oculist and detecting and treating eye diseases in good time. His duties also include regular work in health education so as to make the public aware of the causes of blindness and the measures for its prevention.

Today all medical workers are faced with the great problems of complete eradication of trachoma, control of glaucoma, which is the major cause of blindness, prevention and treatment of eye injuries, prophylaxis of myopia in schoolchildren, etc. The contribution of the doctor's assistant to the treatment and prevention of eye diseases is an important factor in their control.

1

OUTLINE OF THE ANATOMY OF THE ORGAN OF VISION

All parts of the human eye, except the crystalline lens, have developed from the diencephalon and for this reason can be regarded as a *peripheral portion of the brain.*

The organ of vision consists of the *eyeball*, the *protective* structures (orbit and eyelids) and appendages (lacrimal and motor apparatus), nervous connections and nerve centres.

PROTECTIVE STRUCTURES AND APPENDAGES OF THE EYE

The orbit is bounded by four bony walls, and is shaped like a pyramid with its apex directed into the cranial cavity and its open base facing anteriorly. The depth of the orbit is about 5 cm (Figs. 1, 2). The roof of the orbit is formed by the frontal bone in front and the lesser wing of the sphenoid bone behind; the external wall—by the zygomatic and frontal bones and the greater wing of the sphenoid bone; the internal wall—by the lacrimal bone, the anterior portion of the sphenoid bone and the papyraceous lamina of the ethmoid bone; the floor—by the maxilla, the zygomatic process of the molar bone and the orbital process of the palate bone. At the apex of the orbit, in the lesser wing of the sphenoid bone, is the optic foramen (foramen opticum) through which the ophthalmic artery (arteria ophthalmica) enters the orbital cavity; the optic nerve (nervus opticus) passes into the cranial cavity through the same opening. The remaining nerves—oculomotor, abducens, trochlear, the ophthalmic branch of the trigeminal nerve and the superior ophthalmic vein pass through the superior orbital fissure. The latter is formed by an incomplete union of the wings of the sphenoid bone. Externally

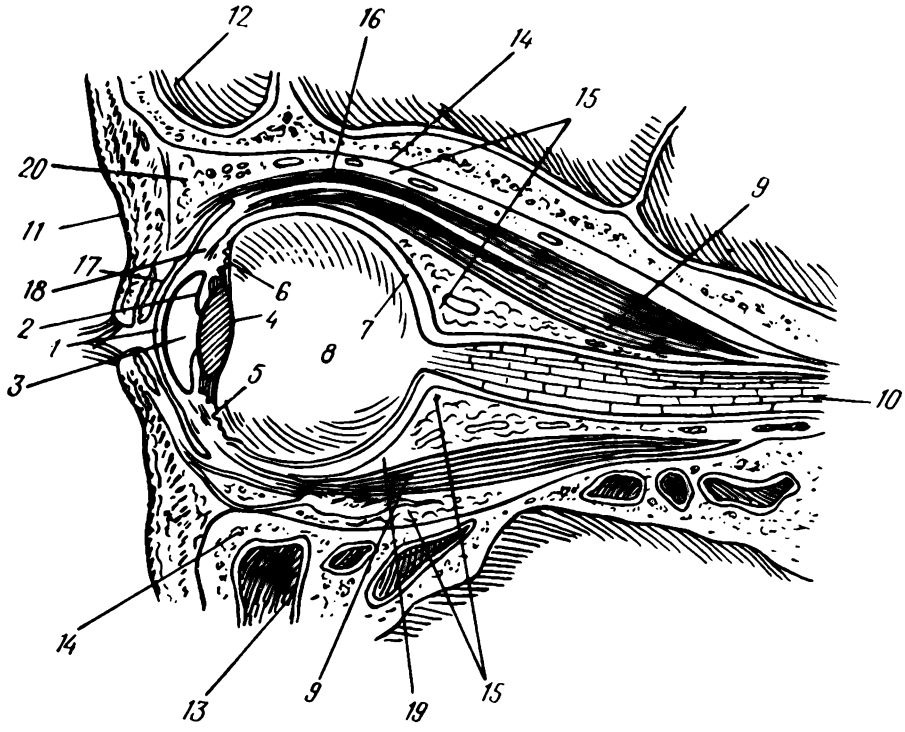

Fig. 1. Saggital section through the orbit and orbital contents
1—cornea; *2*—iris; *3*—anterior chamber; *4*—crystalline lens; *5*—ciliary body; 6—Zinn's ligament; *7*—sclera; *8*—vitreous body; *9*—eye muscles; *10*—optic nerve; *11*—lids; *12*, *13*—paranasal sinuses; *14*—orbital bones and orbital fascia; *15*—adipose tissue; *16*—superior palpebral levator; *17*—palpebral conjunctiva; *18*—bulbar conjunctiva; *19*—Tenon's fascia; *20*—tarso-orbital fascia

and below the orbit lies the *inferior orbital fissure* which carries a branch of the maxillary nerve into the orbit. The orbit is lined with periosteum. At the superoexternal angle of the orbit there is a depression lodging the lacrimal gland; in the inner wall is a niche for the lacrimal sac, which is continuous with the nasolacrimal canal. At the superointernal angle of the orbit there is a bony prominence (trochlear-pulley) over which is passed the tendon of the superior oblique muscle.

The eyeball occupies only the anterior part of the orbit, the remaining space being filled with fat and cellular tissue, muscles, nerves, vessels, ligaments and connective tissue (see Fig. 1). The eyeball is separated from the orbital contents by *Tenon's fascia* (or fascia bulbi), which surrounds the posterior portion of the eyeball. The fascia (or capsule) covers the muscles and together with them is attached to the eyeball and the orbital walls. Anteriorly, the orbit is covered by the eyelids, which together with the *tarso-orbital fascia* (see Fig. 1) adjoining their cartilage form a dense plate protecting the front part of the eye.

The dense inferior and superior orbital margins protect the eyeball from injury. The orbit borders on the frontal, ethmoid and

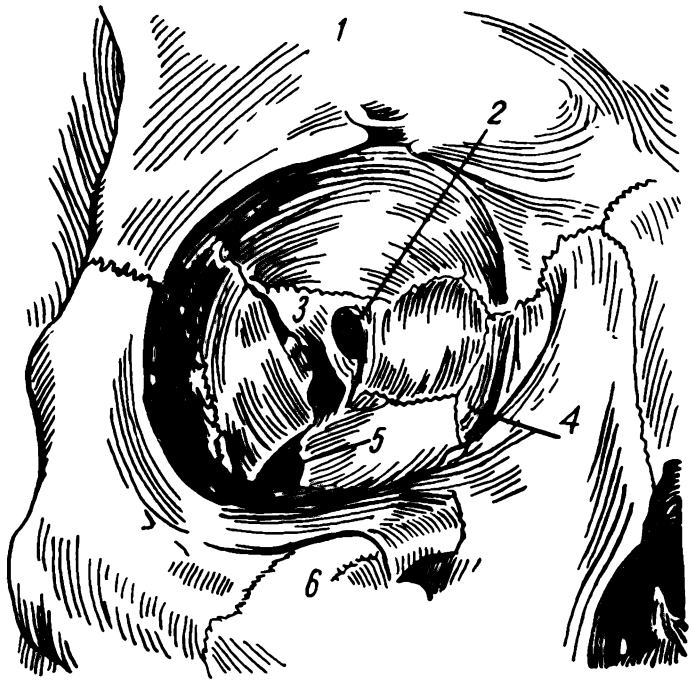

Fig. 2. Bony walls of the orbit. Front view

1—frontal bone; *2*—optic nerve canal; *3*—superior ophthalmic fissure; *4*—lacrimal fossa; *5*—inferior palpebral fissure; *6*—maxilla

maxillary paranasal sinuses; the roof of the orbit separates it from the cranial cavity. This position of the orbit is highly important in the pathology of the eye. For example, it allows an inflammatory process or tumour to extend from the paranasal sinuses to the orbital walls and contents. An injury to the superior orbital roof may involve trauma of the frontal lobe of the brain. As the vascular network of the orbit, particularly the veins, is connected with the veins of the face and brain there is a danger of infection spreading along the facial and orbital veins into the orbital cavity and brain. As a result of this, diseases of the orbit can often cause blindness and even death.

The eyelids (palpebrae) are two skin and muscular folds joined at an angle at the nose medially (inner commissure) and laterally (outer commissure). The eyelids protect the eye during sleep and guard them against bright light, dust and injuries. Winking removes small foreign bodies from the eye and moistens the eyeball surface uniformly. At the inner angle of the palpebral fissure (inner canthus) there is a small eminence, the *lacrimal caruncle*, which is limited by the semilunar fold of the conjunctiva. This is the site of the *lacrimal lake*. The palpebral skin is very thin,

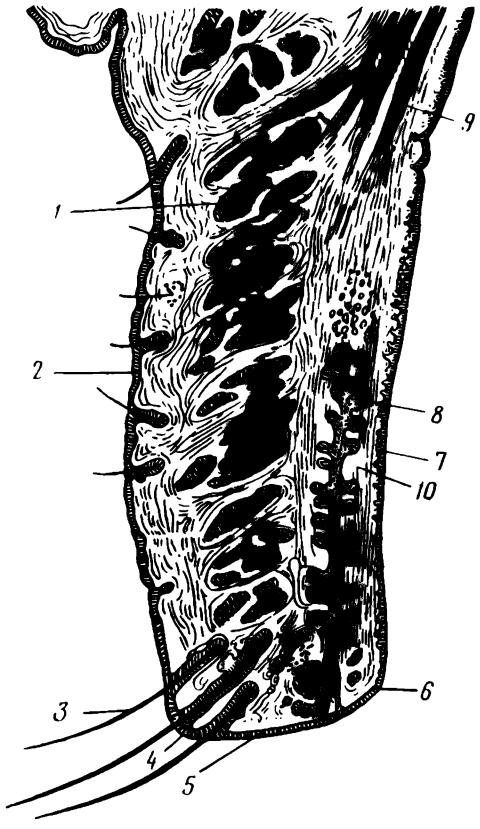

Fig. 3. Saggital section through upper lid

1—orbicular muscle; *2*—lid skin; *3*—lashes; *4*—anterior margin of the lid; *5*—intermarginal space; *6*—posterior margin of the lid; *7*—mucous membrane (conjunctiva) of the lid; *8*—meibomian gland; *9*—superior palpebral levator; *10*—tarsal cartilage

delicate and almost devoid of fat and cellular tissue (Fig. 3). Under the skin lies the *musculus orbicularis oculi* which takes its origin from the inner canthus, describes a circle and joins the inner and outer palpebral commissures and the orbital margins. This muscle closes the eyes, and is innervated by the facial nerve (nervus facialis).

When there is paralysis of this nerve, the eyelids cannot be closed, and the palpebral fissure gapes (*lagophthalmos*). Disturbances in the form and function of the eyelids, particularly those affecting their closure, cause dryness of the mucosal and corneal membranes, which may result in a grave disease leading to blindness.

Behind the musculus orbicularis oculi lies the tarsal cartilage, a dense convex plate of connective tissue. Inside it are embedded the *meibomian glands*. These glands produce a sebaceous secretion which is deposited on the palpebral margins and helps the eyelids to adhere firmly to each other and to the eyeball, and also prevents tears from overflowing the palpebral margin. The cartilage is found only at the ciliary margin. The margins of the cartilages of both eyelids are connected with the orbital margins through the tarso-orbital fascia. The muscle which *raises the upper eyelid* (musculus levator palpebrae) is attached to the upper margin of the tarsal cartilage, the palpebral mucosa and the skin. It is innervated by a branch of the oculomotor nerve. Paralysis of this nerve causes *drooping of the upper eyelid* (ptosis).

The palpebral margin has *anterior* and *posterior limbuses* with an intermarginal space between them. At the anterior border of the lid there are 2 to 3 rows of cilia near which lie the sebaceous glands. The outlet ducts of modified sudoriferous glands open out into the hair follicles of the cilia.

The eyelid vessels originating from the system of the ophthalmic and facial arteries form the superior and the inferior arches. The arteries are accompanied by veins some of which carry blood into

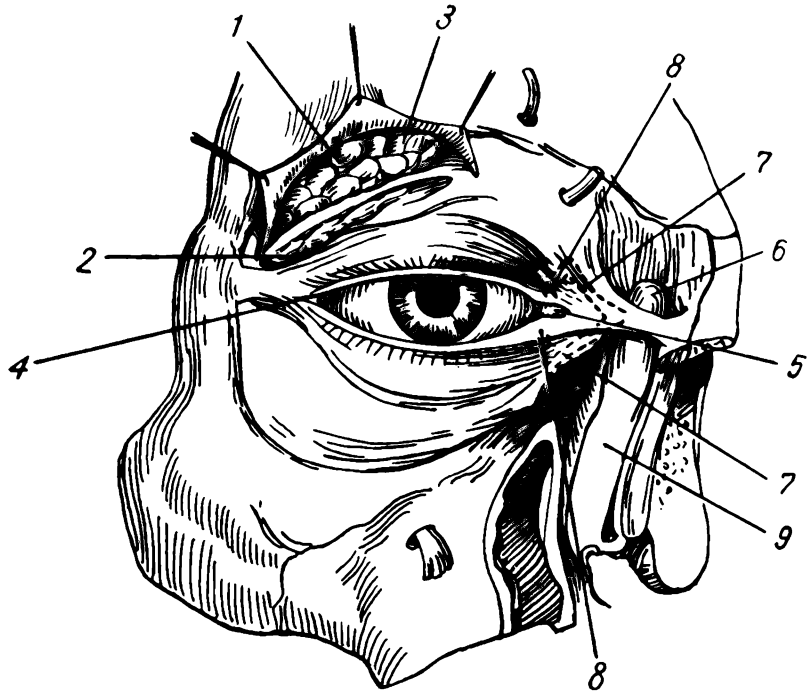

Fig. 4. Lacrimal apparatus

1—lacrimal gland; *2*—palpebral part of the lacrimal gland; *3*—orbital part of the lacrimal gland; *4*—external palpebral commissure; 5—internal palpebral commissure; *6*—lacrimal sac; *7*—lacrimal canaliculus; *8*—lacrimal puncta; *9*—nasolacrimal duct

the cranial cavity and the cavernous sinus (sinus cavernosus), and some of which carry blood into the facial veins. The eyelids need a copious arterial supply for regenerative processes after palpebral injuries. The presence of anastomoses between the veins of the orbit, brain and face may cause infection to spread from the facial and palpebral skin to the orbit and the cavernous sinus. This is facilitated by the absence of valves in these veins, which enables the blood to circulate in various directions.

The insides of the eyelids are lined with *a mucous membrane, the palpebral conjunctiva*, which is reflected upon the eyeball as the ocular conjunctiva extending to the corneal border—the limbus cornea. The superficial layers of the cornea are an extension of the conjunctiva. The place where it is reflected from the eyelid onto the eyeball is known as the conjunctival fornix or the palpebral fold. When the eyelids are closed the conjunctiva forms a closed cavity, *the conjunctival sac*, which is a narrow fissure between the eyelids and the eyeball. The palpebral mucosa is lined with cylindrical epithelium with goblet cells which secrete mucus to moisten the conjunctiva. The latter adheres to the tarsal cartilage and the meibomian glands can normally be seen through it. In the palpebral fold and the ocular conjunctiva the epithelium is made of cuboid

cells with a connective tissue stroma, particularly well-developed in the palpebral fold, which gives the eyeball greater mobility. The mucous membrane has a large number of vessels, the pericorneal region being supplied through the vessels of the eyeball itself—the anterior ciliary arteries—and the remaining part of the membrane being supplied through the palpebral vessels. The conjunctiva is extremely sensitive and when touched generates a defensive reflex—closure of the eyelids. It is innervated by the first and second branches of the trigeminal nerve.

Among the appendages of the eye is *the lacrimal apparatus* which is composed of *the lacrimal gland* and *lacrimal passages* (Fig. 4). The lacrimal gland (glandula lacrimalis) lodged in the bony depression of the supero-external wall of the orbit consists of orbital and palpebral parts. The latter is visible when the upper eyelid is everted, so that the eyeball is drawn sharply inwards and downwards. The outlet ducts of the gland open out into the superior fornix which contains scattered accessory lacrimal glands. The glands secrete about 1 ml of lacrimal fluid daily containing up to 99 per cent water, a certain amount of protein, about 1 per cent sodium chloride, and a bactericidal substance called *lysozyme*, which is an antibiotic of animal origin. The tears moisten the eyeball surface and cleanse the conjunctival sac from microbes, dust and foreign bodies.

The lacrimal passages are made up of the following parts. On the margins of both eyelids and near the inner commissure there are protuberances, *the lacrimal papillae*, with *the lacrimal puncta* which give origin to *the lacrimal canaliculi*. The canaliculi run vertically over a length of 1.5 mm, and then run horizontally and enter the lacrimal sac lying in the lacrimal fossa on the inner wall of the nose. The superior, blind end of the sac is situated a little above the inner palpebral commissure; its inferior end joins the nasolacrimal duct opening out under the inferior nasal turbinate.

The tear drainage mechanism is as follows: the lacrimal fluid from the superior fornix flows down to the inferior fornix under the force of gravity, helped by blinking of the eyelids. The latter facilitate the flow of the lacrimal fluid along the rivus lacrimalis, the space between the eyeball and the lower eyelid, to the inner angle of the palpebral fissure—the lacrimal lake. The fluid flows through the lacrimal puncta and enters the canaliculi, the sac, and the nasolacrimal duct and flows into the nose through the inferior meatus of the nose. When the eyelids are closed the musculus orbicularis oculi attached to the anterior wall of the lacrimal sac retracts and expands the sac cavity, producing a negative pressure in it. Simultaneously the canaliculi expand, and the fluid is drawn from the lake into the sac. When the eyelids are opened its elastic walls shrink and the fluid enters the nasolacrimal duct.

The eyeball has a diameter of about 24 mm, and is nearly spherical in shape (Fig. 5); its anterior portion is more convex.

The external membrane (tunica fibrosa oculi) of the eyeball is formed by a dense opaque white coat called *the sclera* or tunica albuginea and by a convex transparent coat called *the cornea*. The external membrane gives the eyeball its shape. *The sclera*, which makes up five-sixths of the external membrane, consists of dense fibrous connective tissue continuous with the cornea in front. The edge of the cornea at its junction with the sclera is called the limbus. On the inside it faces the angle of the anterior chamber housing a circular venous sinus known as *Schlemm's canal*, which is connected by thin venous branches with the anterior ciliary veins. This is the basic route by which fluid drains from the eye. Posteriorly, the sclera is continuous with the hard sheath of the optic nerve. At the place where the nerve leaves the eyeball the sclera is thinner, and is shaped as a cribriform lamina whose openings convey the nerve fibres of the optic nerve from the eyeball into the orbit. At the posterior pole of the eyeball the sclera is pierced by the short and long posterior ciliary arteries, at the equator by the vorticose veins, and near the limbus by the anterior ciliary arteries and veins. Between the inner layers of the sclera and the vascular tunic there is a narrow capillary fissure—the suprachoroidal space through which the intra-ocular fluids are also drained.

The cornea. The average vertical diameter of the cornea is 11 mm, and the horizontal diameter 12 mm, its depth along the periphery being 1.2 mm, and in the centre 0.8 mm; the cornea is transparent and allows light to pass into the eye since it is without vessels and the myelin sheath on the sensory branches of the trigeminal nerve. The latter form superficial nerve plexuses ending between the epithelial cells with button-like swellings and giving the cornea high sensitivity. The corneal surface is lustrous like a mirror, and is continuous with the mucous membrane (conjunctiva), the sclera and the uveal tract, which is one of the reasons why it is often involved in the pathological processes arising in the above coats.

Since there are no vessels in the cornea it receives nourishment from the looped blood vessels at its periphery and the aqueous humor of the anterior chamber.

The absence of a vascular network accounts for certain features characteristic of the course of corneal diseases. The cornea is a protective coat letting rays of light pass into the eyeball, and is the ocular medium with the greatest refractive power—42.0-43.0 diopters (see below).

The inner surface of the sclera is lined with a vascular membrane, a part of *the uveal tract* (uvea) made up of a pigmented circular

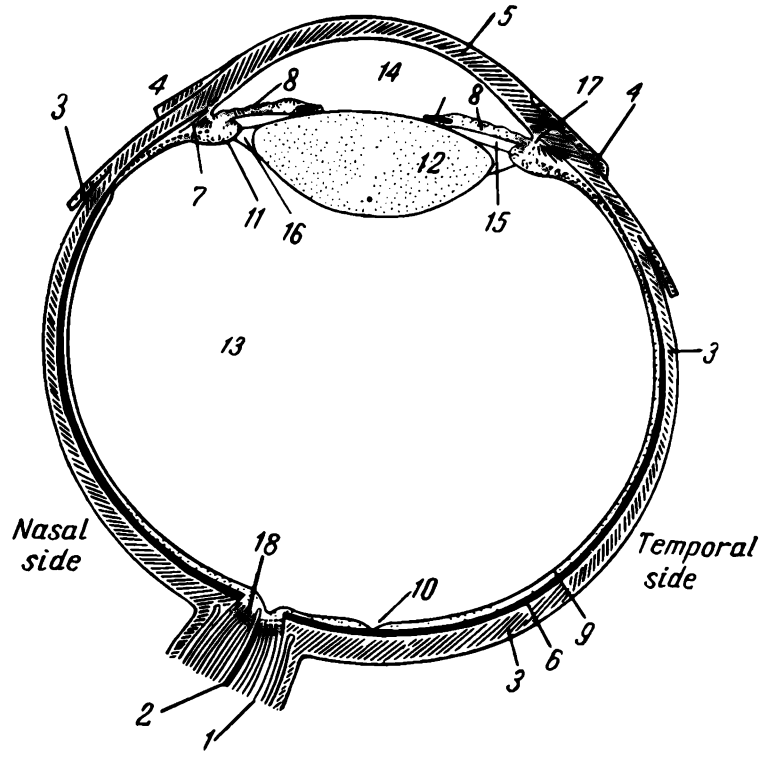

Fig. 5. Horizontal section of the eye

1—optic nerve; *2*—blood vessels in the optic nerve; *3*—sclera; *4*—scleral conjunctiva; 5—cornea; *6*—choroid; *7*—ciliary body; *8*—iris; *9*—retina; *10*—central retinal fossa; *11*—serrated margin of the ciliary body; *12*—crystalline lens; *13*—vitreous body; *14*—anterior chamber; *15*—posterior chamber; *16*—Zinn's ligament; *17*—limbus; *18*—cribriform lamina

membrane known as *the iris*, *the ciliary body* (corpus ciliare), and *the vascular tunic proper*, choroid (chorioidea).

The iris is seen through the cornea. It acts like a septum, separating the anterior chamber (space between cornea and iris) from the posterior chamber (space between iris and crystalline lens). The iris has a central perforation, *the pupil* (pupilla), which changes in breadth, regulating the passage of light into the eye. This is the function of two muscles of the iris; *the sphincter pupillae* which constricts the pupil and *the dilatator pupillae* which dilates it. The former surrounds the pupil as a narrow zone of circular fibres, and is supplied by the oculomotor nerve. The dilatator pupillae is a radial band of fibres extending from the pupil to the root of the iris, radix iridis, and is supplied by the sympathetic nerve. The colour of the iris depends on the amount of pigment contained in it, which determines the colour of the eyes.

The iris is continuous with *the ciliary body* whose longitudinal cross section is shaped like a triangle with rounded sides and angles.

The thickened anterior part of the ciliary body gives off 70 processes and then becomes flattened. The ciliary body lodges the cil-

iary muscle (musculus ciliaris), which is supplied by the oculomotor nerve and takes part in accommodation. The flat part of the ciliary body is continuous with the vascular tunic proper (choroid) which is made up of a connective tissue stroma and three layers of vessels of varying calibre. The vascular tunic is continuous with the sclera only at two points: near the optic nerve and at its junction with the ciliary body. The remaining part of the choroid is in close contact with the sclera because of the pressure of the vitreous body.

Along the whole of its length the uveal tract contains a large amount of pigment which protects the retina from rays of light falling upon the sclera. Thus only those rays which pass through the pupil reach the retina; this prevents diffusion of light and contributes to the formation of a distinct image of the object looked at. The pigmentary epithelium produces visual purple which is contained in the retinal rods. Pigment insufficiency in the eye (albinism) causes a red reflex in the pupil and the entire sclera; the light passing into the eye is diffused. This is one of the causes of weak vision with a blurred image in albinos.

The iris and the ciliary body have an extensive vascular network made up of the anterior and long posterior ciliary vessels, in the choroid, the short posterior ciliary arteries. The vascular networks are interconnected by short two-way branches. Because of this system of blood circulation, diseases of the iris and ciliary body may occur irrespective of those of the choroid. The iris and the ciliary body are pierced by a large number of sensory branches of the trigeminal nerve, whereas in the choroid they are absent. This accounts for the sharp pains felt in inflammatory diseases of the anterior portion of the uveal tract and for their absence in infections of the choroid.

The functions of the uveal tract are as follows: formation of the aqueous humor, regulation of the intra-ocular tension, nourishment of the eye, participation in the act of vision, and accommodation.

The innermost membrane of the eye, *the retina*, lines the entire uveal tract. The optic part of the retina begins at the posterior pole of the eye and terminates at the so-called *ora serrata*, the jagged anterior margin, at the junction of the choroid with the ciliary body. Here, as well as at the optic disc the retina adheres to the surrounding tissue; the remainder of it only adjoins the choroid, and is kept in position by the pressure of the vitreous body.

The retina plays the principal role in the visual functions; it is photosensitive, i.e., sensitive to the rays of light entering the eyeball. It is a thin transparent film comprised of ten layers of nerve cells, their processes and connective tissue. The basic elements of the retina (Fig. 6) are as follows: a layer of rods and cones, which

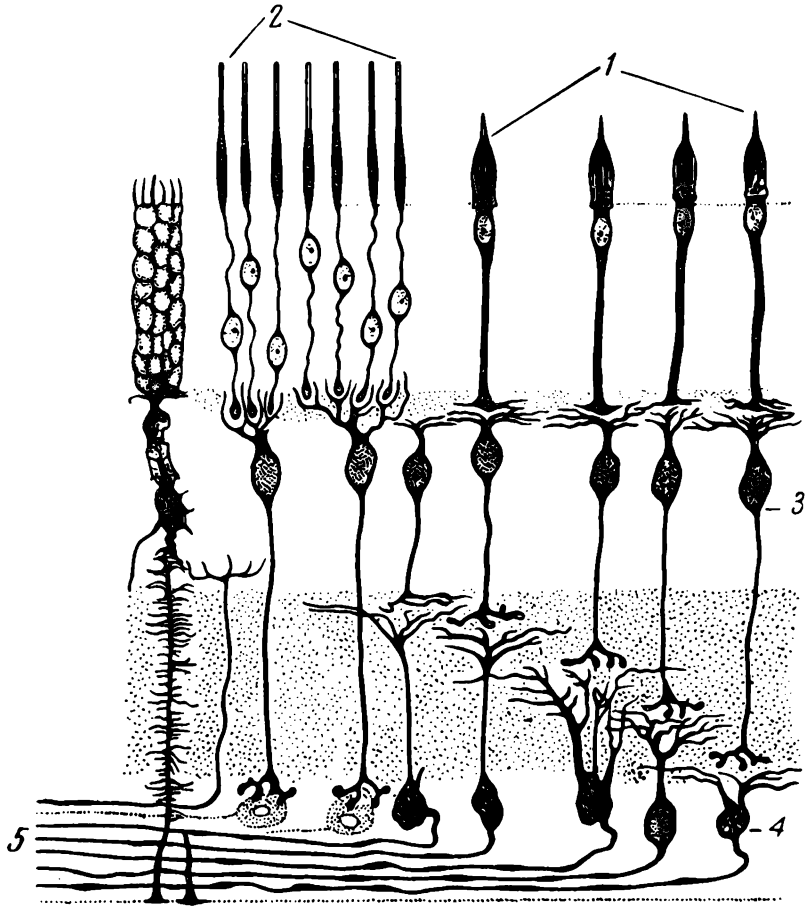

Fig. 6. Schematic view of retinal structure
1—cones; *2*—rods; *3*—layer of bipolar cells; *4*—layer of ganglion cells; *5*—fibres of ganglion cells joining to make the optic nerve

are the photosensitive cells of the optic analysor, followed by layers of bipolar and ganglion cells. The rods and cones lie outside the above-mentioned cells; they adjoin the pigmentary epithelium of the choroid and constitute the neuroepithelial layer of the retina. The other inner layers are called the *cerebral* layer of the retina: its cells are interconnected by nerve fibres.

The retina is supplied with blood from its central artery (Fig. 7) while regeneration of the visual substances required for the continuous functioning of the retina is carried out through the capillary network of the choroid. The structure of the retina is not uniform all over. At the place where the optic nerve leaves the eyeball, in the region of the optic disc, there are no rods and cones and the visual functions are absent. This point corresponds to the so-called blind spot in the field of vision. At a distance of 3-4 mm away from the optic disc is *the yellow spot* (macula lutea) with a small central depression known as *the fovea centralis* containing cones alone. Over the rest of the retina in the direction of the periphery the num-

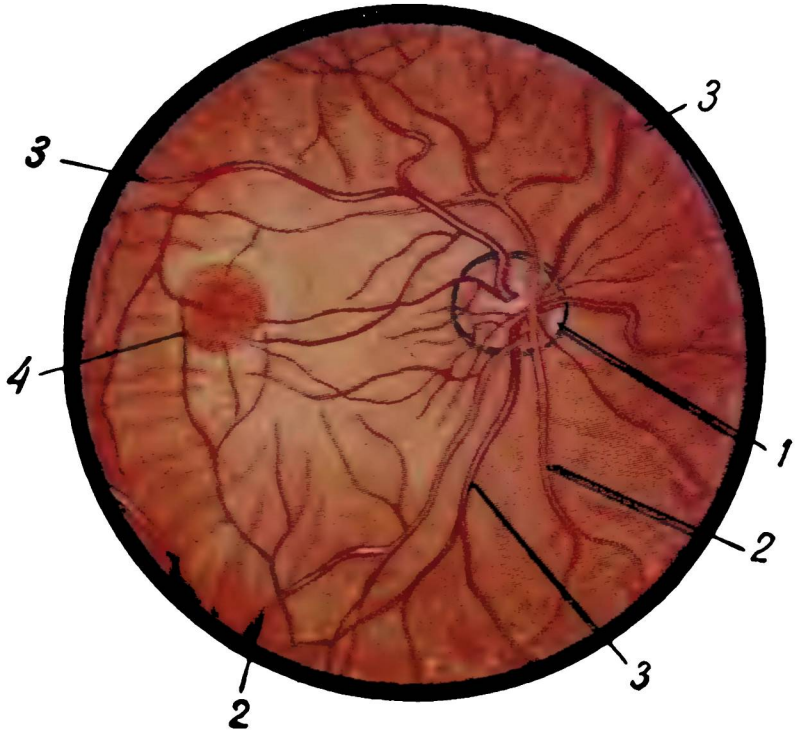

Fig. 7. Normal ocular fundus

1—optic papilla; *2*—macula lutea; *3*—artery; *4*—vein

ber of cones gradually lessens while the number of the rods increases. *The macula lutea is the point of the most distinct and clearest vision called central vision.*

The optic nerve is formed by the fibres of ganglion cells of the retina, and leaves the eye through the cribriform plate of the sclera. A part of it, inside the eye and known as the optic papilla or optic disc, is situated slightly further in than the posterior pole of the eyeball. It is pale pink and oval is shape, with a diameter of 1.5-1.8 mm. Its centre is pierced by the ramified central artery and the retinal vein (Fig. 7).

The anatomo-physiological relationships between the choroid, retina, and optic nerve often account for their being simultaneously involved in a pathological process, regardless of its initial site.

In the orbit, the nerve is made up of the same membranes as the brain; it is shaped like the letter S, which prevents its being strained by movement of the eyeball. After entering the cranial cavity through the bony orbital canal the inner fibres of the optic nerves cross in the region of the Turkish saddle (sella turcica) forming the chiasma opticum; the external fibres do not cross (Fig. 8). Behind the chiasm the nerve continues as *the optic tract* (tractus opticus) in the brain formations, the subcortical region, the primary visual centres of the brain, the external geniculate body (pregeniculum), the thalamus and the anterior pair or the corpora quadrigemina (nates of brain). From the external geniculate body the optic tract passes via the internal capsule towards *the visual centres* in the occipital lobes of the brain cortex.

The transparent contents of the eyeball consist of the aqueous humor, the crystalline lens, and the vitreous body.

The aqueous humor fills the anterior and posterior chambers of the eye. It is very important for nourishment of the eye and for maintaining a constant intra-ocular tension. The aqueous humor forms in the iris and the ciliary body. It passes from the posterior chamber through the pupil into the anterior chamber, at whose angle it is filtered into Schlemm's canal through the slit-like openings in the iridal *pectinate* ligament covering the chamber angle. From Schlemm's canal the humor passes into the anterior ciliary veins, i.e., it enters the general blood stream. Rays of light pass through the aqueous humor with negligible refraction, which can be practically disregarded.

The crystalline lens, which lies behind the iris in a depression in the vitreous body (corpus vitrium) (see Fig. 5), is shaped like a biconvex transparent lentil. It consists of separate fibres arranged in layers one on top of the other. The crystalline lens is contained in a capsule whose anterior surface is thicker than the posterior one and is coated with a layer of cuboid epithelium which produces

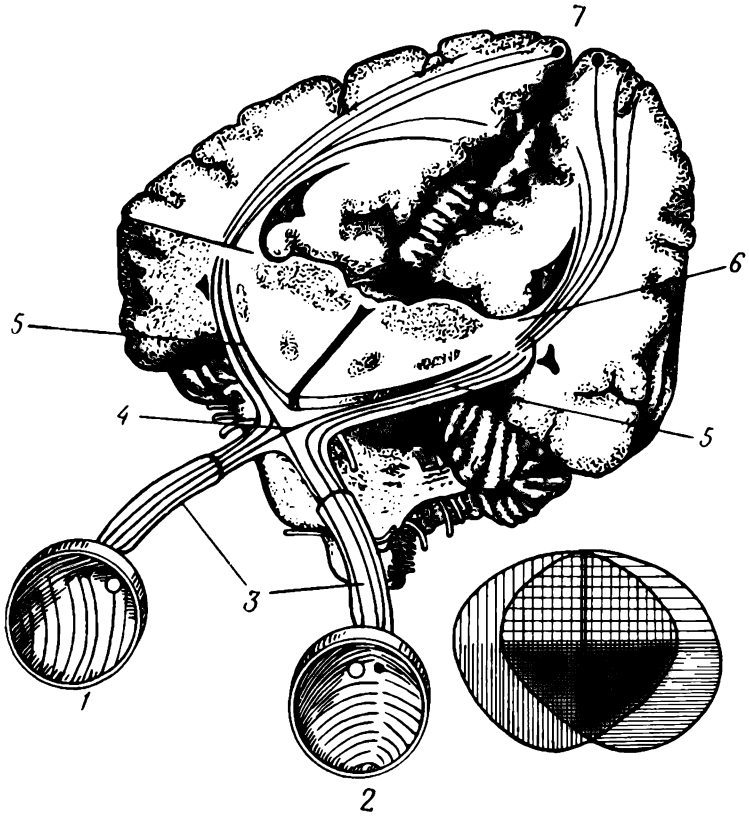

Fig. 8. Schematic view of optic pathways
1—left eye; *2*—right eye; *3*—optic nerve; *4*—chiasm; *5*—optic tract in the subcortex; *6*—external geniculate body; *7*—optic centre in the cortex of the occipital region of the brain

new fibres. The crystalline lens grows throughout life. The newly-formed fibres stratifying under the capsule gradually displace the older fibres toward the centre. The latter fibres thicken, become sclerosed and turn into a nucleus retaining its transparency; the young, softer fibres are arranged along the periphery of the crystalline lens. The substance of the crystalline lens has elastic properties which enable its curvature and, consequently, its optic power to change. As sclerosis of the crystalline lens nucleus advances due to ageing this capacity gradually weakens and finally disappears. The crystalline lens is attached to the ciliary body by means of the ligaments intertwined with its capsule along the equator (see Fig. 5).

The greater part of the eyeball is filled with the transparent gelatinous *vitreous humor* (see Fig. 1). It is pierced by minute fibrils and contains an insignificant number of wandering cells. Vitreous humor contains 98.5 per cent water and 1.5 per cent solid residue.

The crystalline lens and the vitreous humor, like the cornea, have no vessels, and the aqueous humor plays an important role in their nutrition.

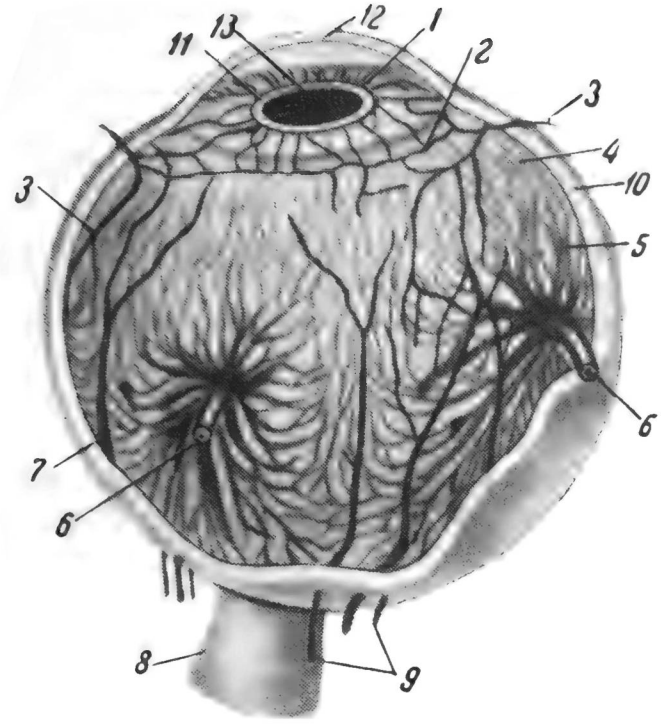

Fig. 9. Vessels of the uveal tract

1—smaller and (*2*) larger system of blood circulation in the iris; *3*—anterior ciliary arteries; *4*—region of the ciliary body; *5*—choroid; *6*—vorticose veins; *7*—long posterior ciliary artery; *8*—optic nerve; *9*—short posterior ciliary arteries; *10*—sclera; *11*—iris; *12*—cornea; *13*—pupil

The cornea, aqueous humor, crystalline lens and vitreous humor constitute *the ocular media*, refracting rays of light admitted through them into the eye (though part is absorbed and reflected). The most powerful refractive medium is the cornea, and then comes the crystalline lens. The vitreous humor and aqueous humor hardly refract light at all. Owing to its capacity to change its refracting power the crystalline lens plays the major role in accommodation of the eye.

The eyeball and its appendages are supplied with blood by *the ophthalmic artery* which takes its origin from the internal carotid artery in the cranial cavity. In addition to the vessels leading to the muscles, eyelids and lacrimal organs, the eyeball receives the central retinal artery which passes inside the optic nerve, and the posterior (long and short) and anterior ciliary arteries which branch within the uveal tract (Fig. 9).

Blood drains from the eyeball through the veins accompanying the arteries, and through the vorticose veins (venae vorticosae) which pierce the sclera; the blood drains into the ophthalmic veins and from these into the cavernous sinus (sinus cavernosus) lying at the Turkish saddle (sella turcica). The venous network of the orbit

communicates with that of the face. These anastomoses are responsible for inflammatory processes spreading from the facial skin, particularly from the region of the upper lip, nose, and forehead, to the orbit and from there to the brain in the case of cavernous sinus thrombosis.

MUSCLES OF THE EYE

The eyeball is moved by the following six muscles: the internal, external, superior and inferior rectus muscles and the superior and inferior obliquus muscles (see Fig. 1).

These muscles, except for the inferior obliquus muscle, take their origin from a common tendinous ring (Zinn's ligament) at the optic foramen of the orbit from where they diverge in a fan-like manner and pass forward, to the eyeball. The rectus muscles pierce the capsule of Tenon and are attached by tendons to the sclera at different distances from the limbus (5.5-7.5 mm). The superior obliquus muscle passes across the superointernal wall of the orbit, runs with its tendon over the trochlear pulley and is attached to the eyeball behind the equator. The inferior obliquus muscle takes its origin from the orbital periosteum, at its inferointernal margin and, bypassing the eyeball, runs under the external rectus muscle and is attached to the eyeball behind the equator. The external rectus muscle is supplied by the abducens nerve, the superior obliquus muscle by the trochlear nerve, and the remaining muscles by the oculomotor nerve. The rectus muscles rotate the eyeball in the directions corresponding to their positions; the external muscle—only outward, the internal—inward, the superior and inferior rectus muscles—upward and downward and, in addition, they deflect the eyeball inward. The obliquus muscles draw the eyeball outward, the superior muscle simultaneously rotating the eyeball downward and the inferior muscle upward.

It can be seen that these muscles are subdivided into four groups according to their physiological functions: (1) *the abductor muscles*—the external rectus and both oblique muscles; (2) *the adductor muscles*—the internal, superior and inferior rectus muscles; (3) *the levator muscles*—the superior rectus and the inferior oblique muscles; (4) *the depressor muscles*—the inferior rectus and the superior oblique muscles.

The concerted coordinated movements of both eyes, particularly the approximation of the visual axes by fixing the gaze upon an object placed at the near-point (*convergence*), cause an image to be formed in corresponding places on each retina and bring about their coalescence, which is necessary for distinct, clear binocular vision (with both eyes). Any movement of the eyeball is caused by a group of muscles. For example, outward rotation of the eyeball requires

the abductor muscles to be tensed and the adductor muscles to be simultaneously relaxed.

In addition to the vessels and oculomotor nerves mentioned above, the orbit transmits the first branch of the trigeminal nerve (nervus ophthalmicus) which provides the sensory and trophic supply of the eye and its appendages. Deep in the orbit, at a distance of 1 cm from the posterior pole of the eye, is the mixed ciliary nerve ganglion lying on the optic nerve between it and the external rectus muscle. This ganglion is composed of branches of the trigeminal, oculomotor and sympathetic nerves. The ciliary nerves passing out of this ganglion enter the eyeball to form the nervous system of the ciliary body and the iris.

2

THE VISUAL FUNCTIONS AND METHODS OF EXAMINATION

The organ of vision is the most important means of perception of the external world. Man can see by bright light and at dusk, can distinguish the shape and colour of objects, and can orientate himself in space. The organ of vision can perceive negligible light stimuli, adapt itself to illumination of variable intensity, can see objects clearly at different distances, and can merge the images produced on the retina of each eye into a single picture. The mobility of the eyes enables them to observe a vast space. The interconnected visual functions are controlled by the brain cortex and ensure an adequate and correct perception of objects of the external world.

This perception or *the act of vision* takes place in the following way.

The rays of light reflected by the object being looked at pass through the ocular media where they are refracted, and impinge on the retina, causing stimulation of the rods and cones. The effect of the light is to bleach the visual purple and to bring about other processes leading to nerve excitation, which is transmitted to the optic nerve, optic tract, subcortical and cortical centres. It is in the latter that visual sensation is produced. The capacity to see and to analyse these sensations is the result of a number of conditioned reflexes based on the combined action of the optic nerve and the muscular apparatus of the optic analysor.

CENTRAL VISION AND VISUAL ACUITY

If the rays of light (Fig. 10) impinging on the retina converge (focus) on the yellow spot (macula lutea), all the details and colour of the object looked at will be seen clearly. This vision is known as *central vision*. If the image on the retina is formed outside the yel-

low spot, the object will be seen indistinctly and will have its outlines and details blurred. The state of central vision is usually determined by examining *the visual acuity*. The latter is found from the smallest object the eye can see at a given distance; the smaller the object the eye can see at a given distance, or the larger the distance from which this object can be seen, the greater the visual acuity. And conversely, the larger the object and the shorter the distance, the lower the visual acuity. Consequently, the acuity of vision is determined by the correlation between the size of the object viewed and the distance from it.

The average human eye can distinguish the details of an object if the rays of light emitted from two points and refracted in the ocular media form a certain angle known as the *optic* or *visual angle* with a minimum value of 1 min (Fig. 11). This capacity to discern objects is assumed normal, and is conventionally taken as equal to 1.0. The images of two luminescent points will merge into one if the rays of light impinge on one cone or two adjacent cones. A divided image of the points may form if between two light-stimulated cones there is one intermediate cone unstimulated by light. This is possible at an optic angle of not less than 1 min, since this corresponds to the linear value of the diameter of a cone.

Visual acuity is estimated by means of charts with black letters, symbols or drawings (for children) of a definite size, arranged in rows. The charts are so designed that the letter or symbol elements

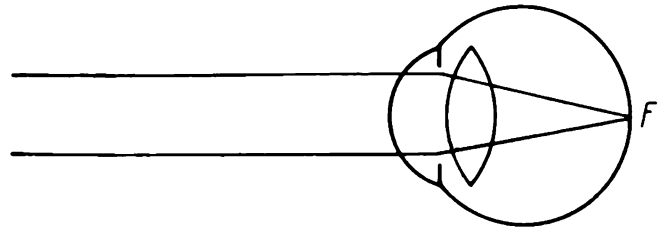

Fig. 10. Light rays converging to a focus on the retina

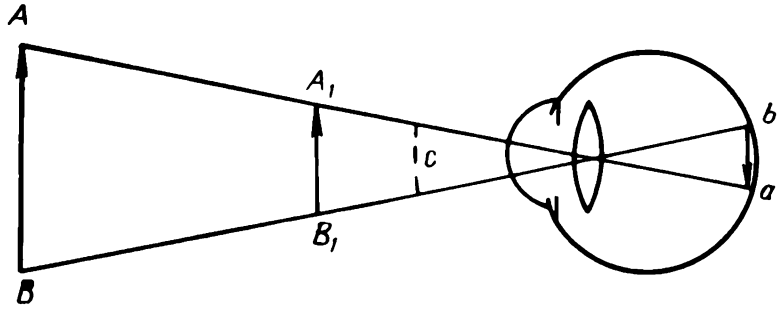

Fig. 11. Formation of an image on the retina

A large object at a great distance from the eye (AB) gives on the retina an image (ab) the same size as that from a smaller object closer to the eye (A_1B_1), C—optic angle

Fig. 12. Examination of visual acuity with the aid of a chart

in the 10th row are visible at a distance of 5 m at an angle of 1 min which, as noted above, corresponds to a visual acuity of 1.0.

The charts are based on the decimal system in which each row differs from the one above by 0.1 of visual acuity. For example, if at a distance of 5 m, a patient can see symbols only in the first row his visual acuity is equal to 0.1; the corresponding figure for the second row is 0.2, for the fifth row 0.5, etc.

The patient's answers should be taken down as follows: Vis OD (right eye) =0.5, Vis OS (left eye) =0.6, or Vis OU (both eyes) - 1.0. The charts should be kept clean. To ensure steady and bright illumination of the charts they are placed in a lighting device (Fig. 12) with mirrors attached to the inner surfaces of its walls for greater uniformity of lighting. A 40W electric bulb screened from the patient with an opaque shield is set in front. It is best to illuminate the charts with daylight lamps (A. V. Roslavtsev).

An examination can be carried out at any time of day with the aid of illuminated charts. The lighting device for the charts is usu-

ally set facing a window. The charts can be protected from fading by covering them with cotton curtains when not in use. The visual acuity is measured at a distance of 5 m. Each eye is examined separately, the eye not being tested being covered with a white plastic or cardboard square 9×12 cm. The examiner points to consecutive symbols on the chart, starting from the top and working downwards, and the patient is told to name them. The visual acuity can be determined from examinations at distances other than 5 m by using the following formula:

$$V = \frac{d}{D}$$

where d is the distance between the patient and the chart, D is the distance at which the normal eye can see symbol elements in a given row and V is the visual acuity; corresponding values are printed on the chart to the left of each row.

For example, if a patient seated at a distance of 4 m from the chart can see symbols only in the first row which normally he would have distinguished from a distance of 50 m, his visual acuity is $\frac{4}{50} = 0.08$ rather than 0.1 as would have been the case if he had seen symbols in the first row at a distance of 5 m. If the patient cannot distinguish symbols even in the first row at a distance of 5 m, he is told to come nearer the chart, and a note is made of the distance at which he can see the first row. Thus, if he can distinguish symbols in the first row only at a distance of 1 m, his visual acuity is $\frac{1}{50}$, i.e., 0.02; at a distance of 2 m the corresponding figure is $\frac{2}{50} = 0.04$, etc. The examiner may hold up his fingers against a black background to be counted, noting the distance at which the patient gives the correct answers. In both cases the above formula is used, assuming that the thickness of the fingers is approximately equal to that of the symbols in the first row. If the patient is unable to see symbols in the first row even from a distance of 1 m and cannot count the fingers shown him at this distance or nearer, the photoreceptive power of the corresponding eye should be examined.

For this examination the patient is seated in a dark room, the light source is placed to the left and behind him, and a light beam from an ophthalmoscope (an instrument for examining the interior of the eye) is directed into each eye separately (see Fig. 23). The examiner inquires whether the patient can see the light and determine its direction. If the patient answers correctly, his vision is considered to be equal to the light sense (designated $\frac{1}{\infty}$) with correct projection. If the patient can see the light but cannot determine its

direction, his vision is equal to the light sense ($\frac{1}{\infty}$) with incorrect projection. Finally, if the patient is completely unable to differentiate between light and darkness the faculty of sight in the eye under examination is absent altogether (i.e., equal to 0).

It is extremely important to determine the presence of photoreception and the state of light projection in order to decide whether surgical interference is necessary. For example, when the cornea or lens is opaque, the acuity of vision may be impaired to such an extent that light can be perceived only in correct projection; this will point to retention of the optic nerve functions. In such cases, there is hope that an operation will be successful and the patient's vision restored. Incorrect light projection usually occurs in disturbances of the retinal and optic nerve functions; an operation for opacity of the cornea or lens will therefore be useless. A visual acuity of zero is evidence of hopeless, incurable blindness.

Determination of the visual acuity is normally the first procedure in examining every new patient. A lowered visual acuity may be the first sign of many eye diseases and, during therapy, a change in visual acuity is often one of the principal signs of improvement or deterioration.

Since the determination of visual acuity is based on the patient's subjective symptoms, it is sometimes necessary to take account of a possible aggravation of partial blindness or simulation (for instance, for disability rating). Cases of dissimulation are also encountered frequently, for example when impaired vision involves the loss of a job requiring high visual acuity (vehicle and engine driving, etc.). In such cases, the actual visual acuity is determined by using B. L. Polyak's control tables with the symbols arranged in a different order, as well as by other methods. In particular, one can perform the examination from different distances and verify the findings by using the visual acuity formula. In this procedure visual acuity will not correspond to a particular row of symbols, as in the usual method. For example, if a patient can distinguish symbols in the second row at a distance of 5 m, his visual acuity is 0.2; at a distance of 2.5 m it is 0.4, and so on. One can also show letters glued upon pasteboard squares. It is difficult for a patient to memorize the relation between letter size and visual acuity, so his answers will be wrong.

COLOUR VISION

Colour vision like visual acuity is a function of central vision, i.e., of the macula lutea. The ability to distinguish colours is very important; colour vision enables man to see coloured signals, the hues of coloured fabrics and the varied colours of nature. Complete

or partial colour-blindness is a hindrance to work in the transport service, the dyeing, chemical and textile industries, in certain medical specialities, etc. The eye is capable of perceiving an infinite variety of colours and hues. All of them appear from the displacement of the seven basic colours of the spectrum—red, orange, yellow, green, blue, indigo and violet—produced by the dispersion of a ray of white light passed through a prism. The principal colours are red, green and violet; in combination they produce all the other colours and hues.

The trichromatic theory of colour vision is based on the work of M. V. Lomonosov, T. Young and H.L.F. Helmholtz corroborated with modern findings. According to this theory, the retina has three sets of fibres which respond to stimulation by sensations of red, green and violet, respectively. Normal colour vision is possible only when colour vision fibres function simultaneously and adequately, since excitation of one of them entails simultaneous slight stimulation of the others. Therefore, complete or partial loss of perception in one set of fibres brings about a change in the perception of all colours. Defective colour vision is known as *anomaly*.

As regards their capacity to see colours all people can be subdivided into those with normal colour vision, those with partial colour blindness and those with total colour blindness.

The above disorders in colour vision are congenital, and are found in approximately 4-6 per cent of men and much less frequently in women. Colour-blind persons, let alone those with partial colour blindness, may be unaware of their defect because in everyday life they may be capable of differentiating colours by their richness, brightness and other indirect signs. Colour vision disorders are usually diagnosed by examination with the aid of special charts based on definite colour combinations.

Y. B. Rabkin's charts consist of varicoloured circles arranged to form a numerical or geometrical figure which can be seen distinctly with normal colour vision. If the colour vision is disturbed, the patient will be unable to see certain figures and instead will see concealed figures invisible to those with normal colour vision. An examination of colour vision is performed in daylight. The patient is seated with his back to the light so that he can see the charts clearly. The charts are shown one by one from a distance of 1 m, for not longer than 10-20 sec each. The patient is first shown the charts used for control and acquaintance; they can be deciphered by the colour-blind also. The other tables are diagnostic ones and help to identify and differentiate between the various types of colour-blindness. Testing of colour sensation is required in selecting applicants for certain jobs and in examining people employed in all the transport services and on other jobs requiring colour vision.

PERIPHERAL VISION AND FIELD OF VISION

Vision with the periphery of the retina is known as *peripheral* vision. It is also called crepuscular vision because it allows the perception of surrounding objects in dim illumination and orientation in space; the aggregate of all points seen with the eye steadily fixed forms *the field of vision.*

The great importance of peripheral vision can be easily realized if an attempt is made to move about a room with narrow tubes (for instance, stethoscopes) set against the eyes to shut out peripheral vision; such movement is very difficult or impossible.

Pathological changes in the peripheral portions of the retina as well as deficiency in vitamin A and diseases of the liver cause *nyctalopia* or *night-blindness.* The sufferer has poor sight at dusk, and at night becomes quite helpless in spite of normal visual acuity in bright light.

Fig. 13. Examination of the visual field with a perimeter

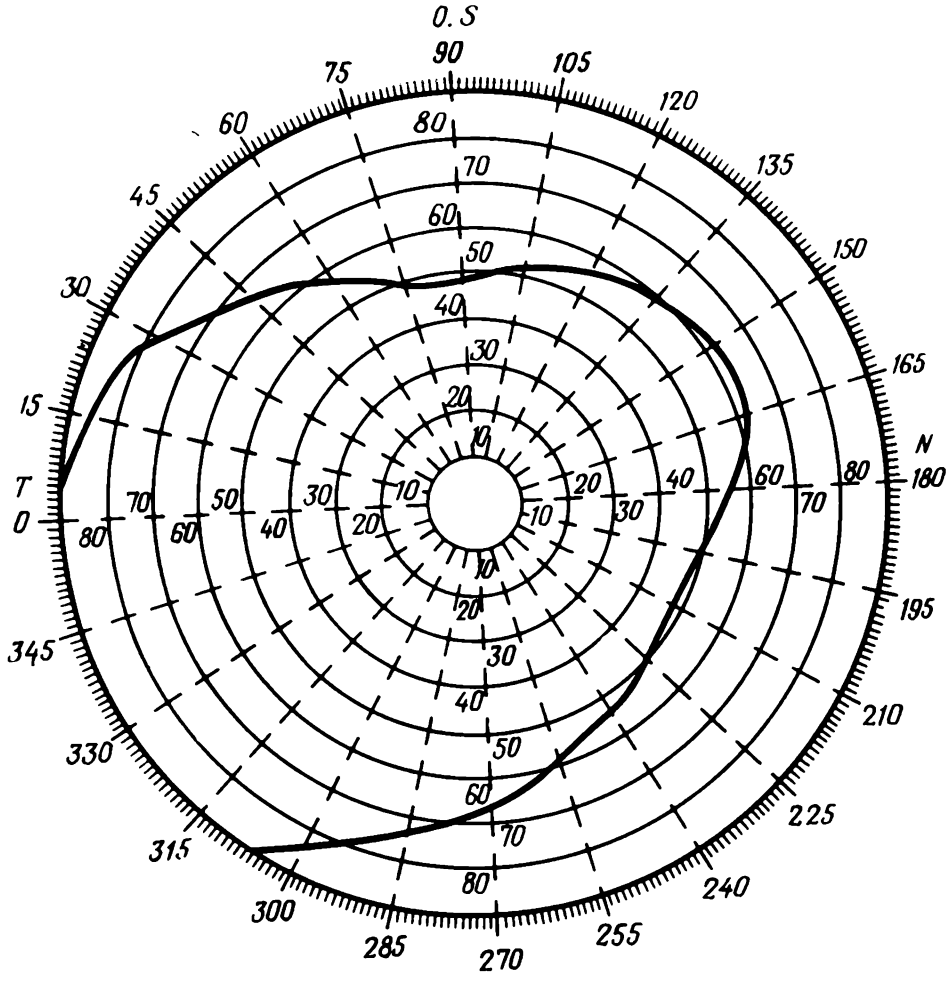

Fig. 14. Limits of the normal visual field in examination with a white object (continuous line)

Disturbances of peripheral vision occur in certain conditions of the retina, the optic nerve, the conduction pathways and the central nervous system.

Peripheral vision is examined by determining the field of vision and the light sense, i.e., the capacity to perceive light of minimum intensity. Examination of the field of vision is a highly important procedure in the study of the functions of the optic nerve apparatus; it often becomes involved both in intra-ocular diseases and, particularly, in diseases of the central nervous system. The field of vision is commonly examined with a perimeter used to determine boundaries and identify defects, if any. The perimeter (Fig. 13) is a black-coloured plate bent in a semicircle and movable on a pivot about its axis. It is graduated in degrees from 0° in the centre to 90° at the periphery, and fixed to an upright.

The position of the plate from the horizontal to the vertical is measured in degrees on a scale attached to the centre of the rear

surface of the plate. During examination, the patient's chin is placed on a support, one eye is covered with a bandage, and the other is fixed on the white disc in the centre of the plate. A white reflection of a ray of light is moved along the perimeter plate from the periphery to the centre. The patient looking at the centre of the plate is told to note the moment when he sees the white object. The position of the object at this moment will correspond to the boundary of the field of vision in a given meridian. The boundaries are determined in the vertical, horizontal and two diagonal (oblique) meridians. The findings obtained in the examination are entered on a chart (Fig. 14) where concentric circles indicate in degrees the distance from the centre at which the object was noted, and the meridians show the position of the plate when the examination was carried out. The same method is used to determine the boundaries of white, indigo, red and green colour vision by means of coloured objects.

The normal boundaries of the field of vision for the colour white (with a 5-10 mm object) are as follows: outwards 90°, inwards 60°, downwards 70°, upwards 60°. The boundaries of the coloured fields of vision are narrower; the widest is the field of vision for the colour indigo, that for the colour red is narrower and that for green is narrower still.

There is also the so-called control method of measuring the field of vision; it is very simple but inaccurate: the boundaries are determined by comparison with those of the examiner. The latter is seated at a distance of 50 cm from the patient who has one eye covered with a bandage. The patient fixes his gaze on the opposite eye of the examiner. Half way between himself and the patient the examiner holds a white object (diameter of circle or side of square 1 cm) on a black rod, or his fingers, and gradually moves them from side to side and up and down. The patient is told to note the moment when the object can no longer be seen, i.e., when it is outside his field of vision. If this occurs simultaneously for the examiner and patient the latter's field of vision is considered to be normal; otherwise it is assumed that the patient has a narrowed field of vision.

The field of vision may be narrowed concentrically (for instance, in pigmentary degeneration of the retina, diseases and atrophy of the optic nerve); focal lesions of the retina, haemorrhages, and inflammation of the optic nerve and conduction pathways may produce blind spots in the visual field known as *scotomata* (sing. scotoma); there are cases of blindness in one half of the visual field—*haemianopsia* (for instance, in lesions of the optic tract, etc.).

Defects in the field of vision can be detected during the determination of its boundaries; the object should be moved from the periphery to the centre of the plate and the patient asked whether the object is lost from the field of vision. Any defects discovered are put on a chart.

PHOTORECEPTION

Photoreceptive power depends on the activity of the entire optic nerve apparatus, from the neuroepithelial layer of the retina to the brain cortex. Photoreception is connected with the photochemical processes of breaking down and regeneration of visual purple in the retina; these processes occur continuously and simultaneously, and depend on the intensity of illumination. Examination of photoreception is very important in certain diseases, such as glaucoma, diseases of the liver, the central nervous system, vitamin deficiency, etc., because this visual function becomes impaired earlier than all others. Photoreception is lost later than the other visual functions. This examination determines the minimum (threshold) light stimulus perceived by the eye. The stimulation threshold is variable and depends on many causes, above all, on the brightness of the initial illumination. After darkness, light at first causes a sensation of blindness; conversely, on entering a dark room from a light place some time elapses before objects can be seen. This ability of the optic analysor to adjust itself to light of different intensities is known as *adaptation*. There is adaptation to light and adaptation to darkness. The most important practically is *dark adaptation*, which is examined by means of a special instrument known as *an adaptometer*.

Persistent, progressive nyctalopia is observed in diseases of the retina, the optic nerve, the central nervous system and the liver. Complete or partial vitamin A deficiency causes temporary disturbances in adaptation, which is quickly cured by the intake of cod-liver oil, vitamins E and A or food rich in them (liver, carrots, butter and wheat, maize, and other oils).

Examination of photoreception is very important not only in various pathological conditions, but also in the selection of persons for work in illumination of variable intensity, in all the transport services, for troops serving in special branches of the services, etc.

A relationship has been established between photoreception and the activity of the brain cortex.

BINOCULAR VISION

If an object is viewed with both eyes a separate image will be formed on the retina of each eye. Normally, however, both images merge into one. Vision with both eyes is known as binocular vision. The images from the right and the left eye coalesce into one.

This perfect type of vision enables man to see the size and contours of objects, their position with regard to one another, and to determine correctly his own position and that of surrounding objects

in space. Binocular vision is possible when the visual axes of both eyes are brought together accurately to the object looked at. This is known as *convergence*. The stimulus for this convergence, effected through a corresponding contraction of the internal recti muscles of the eye, is a reflex desire for fusion of the images formed on the retina of each eye. When the gaze is fixed upon an object placed at the near-point, accommodation is brought into play simultaneously with convergence. Binocular vision is made possible by the combined action of the optic nerve and muscular apparatus.

The outstanding Russian physiologists I. M. Sechenov and I.P. Pavlov made a great contribution to the theory of binocular vision. They proved that the perfect functioning of the optic analysor is based on a chain of conditioned reflexes. Binocular (otherwise called spatial, three-dimensional) vision plays an important role in the life of man. It is especially important in certain jobs (for pilots, specialists in precision mechanics, range-takers, etc.). It is examined by means of special instruments, including the stereoscope. When persons with normal binocular vision view special pictures through a stereoscope objects appear to have solidity and relief.

3

REFRACTION AND ACCOMMODATION

OCULAR REFRACTION. OPTIC LENSES

Normal clear vision depends upon the transparency of the ocular media and the normal functioning of the optic nerve apparatus, i.e., conditions must be such that a distinct image is formed in the region of the macula lutea. This image is produced by rays of light brought to a focus on the retina, which depends not only on the refractive power of the ocular media but also on the length of the eyeball. The correlation of these two values determines the refractive power of the eye, i.e., ocular *refraction*. This correlation determines the variable position of the focal image of an object located at a great distance from the eye (at "infinity"). Parallel rays from this object under different conditions may be brought to a focus upon the retina, in front of or behind it. The position of the focus for parallel rays determines *the type of refraction*: *emmetropia* or *normal* refraction in which parallel rays after refraction are brought to a focus upon the retina; *hypermetropia* or *far-sightedness* in which the focus is behind the retina; and *myopia* or *near-sightedness* in which the focus lies in front of the retina (Fig. 15).

To understand the various types of ocular refraction one has to be familiar with the refractive power of optic lenses because the eye can be likened to a complex optical instrument acting as a convex lens and converging rays of light. *The convex* (converging) *lens* can be imagined as being made up of two prisms with their bases put together; parallel rays of light passing through this medium will be deflected towards the bases of the prisms and become convergent; such a lens is said to be positive and is designated by the sign (+).

The concave (diffracting) *lens** acts like two prisms put together

* Today both lenses are made concavo-convex instead of convexo-convex and concavo-concave.

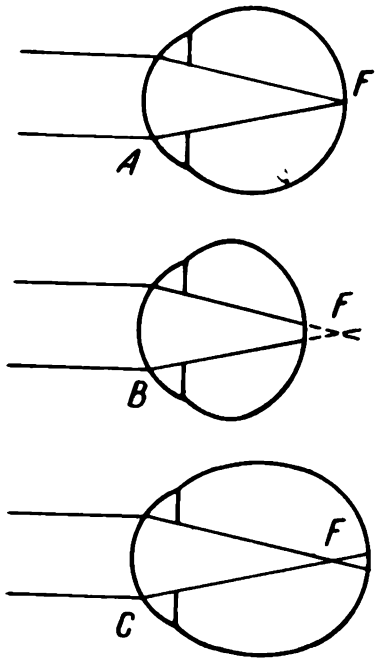

Fig. 15. Position of the focus of parallel rays in the eye for the various types of refraction

A—emmetropia; *B*—hypermetropia; *C*—myopia; *D*—focus of rays of light

at their apexes. Parallel rays refracted in this lens will diverge and the focus (imaginary) will be on the same side of the lens as the object. This lens is said to be negative and is designated by the sign (—). Optic lenses have variable refractive power measured in diopters and depending on the curvature of their surfaces.

A diopter (D) is equal to the refractive power of a lens whose focal length is one metre. The power of the lens is inversely proportional to the focal length; in other words, the shorter the focal length, the greater the power of the lens and, conversely, the longer the focal length the lower the power of the lens.

An eye in which there is correspondence between the power of its optic system and its length is called a commensurate eye, and parallel rays of light are brought to a focus on the retina. Such an eye has normal refraction, i.e., *emmetropia*. Eyes in which commensurability is absent are called *noncommensurate, ametropic* eyes. They are either near- or far-sighted (myopic or hypermetropic) owing to refraction anomalies. A myopic eye may have a longer anteroposterior axis so that parallel rays of light, after refraction in the cornea and lens, are brought to a focus in front of the retina. A hypermetropic eye may have a shorter anteroposterior diameter, and the focus of parallel rays will lie behind the retina. The differences in eyeball sizes in the various kinds of refraction are schematically represented in Fig. 16.

Anomalies of refraction are rarely associated with an increased or decreased refractive power of the eye with a normal axial length. More frequently these anomalies are caused by lack of correspondence between the length of the axis of the eye and the ocular refraction, although these vary within normal limits.

Since in a myopic eye the focus of parallel rays lies in front of the retina, clear vision of distant objects is impossible. At the same time, near objects are clearly visible since diverging rays from them are brought to a focus on the retina of a myopic eye.

In a hypermetropic eye the focus of parallel and diverging rays does not lie upon the retina (it is adapted to the reception of converging rays which are nonexistent in nature). A hypermetropic eye can therefore see distant and near objects clearly only with high-degree accommodation or through optic lenses.

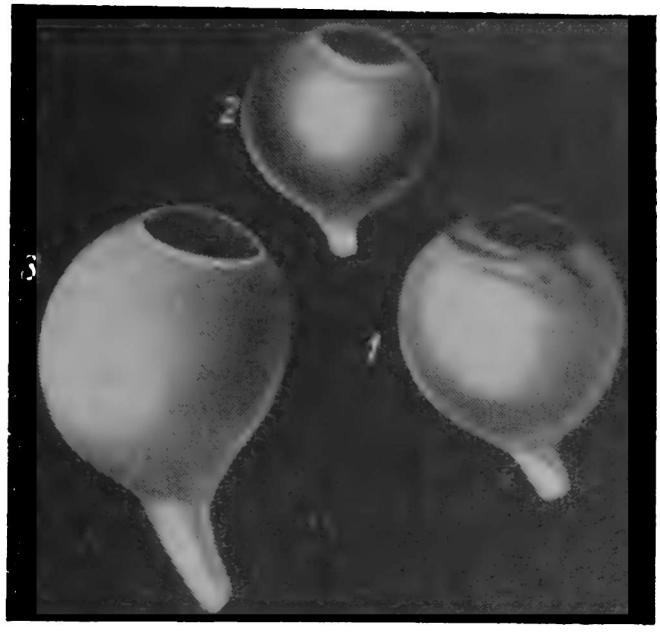

Fig. 16. The size of the eyeball for the various types of refraction
1—emmetropia; *2*—hypermetropia; *3*—myopia

There are also eyes in which rays of light are differently refracted in different planes. Such refraction is known as *astigmatism*. In this condition rays of light are brought to a focus on the retina as a line rather than as a point; clear perception of objects and good vision are impossible without correcting lenses. Astigmatism is known to be of the following three types: *simple* astigmatism when emmetropia is present in one plane of the cornea while in the other plane, there is hypermetropia or myopia; *complex* astigmatism when there is either hypermetropia or myopia in both planes although of different degrees; and *mixed* astigmatism which is a combination of myopia and hypermetropia.

ACCOMMODATION

If ocular refraction could not be changed, the emmetropic eye would be able to see distant objects only and reading would be impossible. A near-sighted person would be able to see only at a distance corresponding to his refractive power; for example, in 5.0 D myopia at a distance of 20 cm, and in 10.0 D myopia at a distance of 10 cm, etc. The hypermetropic eye would not be able to see either distant or near objects clearly. People, however, can usually see clearly objects at different distances from their eyes. This is made possible by *accommodation*, i.e., the capacity of the eye to adapt

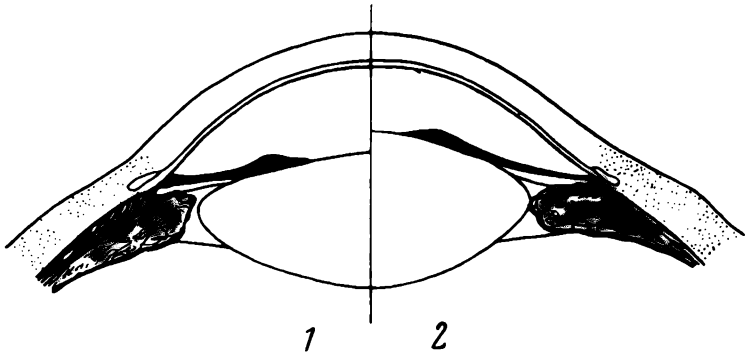

Fig. 17. Diagram of accommodation
1—crystalline lens when accommodation is at rest;
2—crystalline lens during accommodation strain

itself to vision at different distances by an increase or decrease in refraction. This adaptation takes place continuously, and is a conditioned reflex.

The mechanism of accommodation is as follows: under the influence of nerve impulses corresponding to the distance from the object viewed, there is contraction of the ciliary muscle and relaxation of Zinn's ligament fibres and the fibres attached to the crystalline lens capsule. This results in loosening of the capsule, and the lens becomes more convex in shape owing to its elasticity (Fig. 17). This increases its refractive power and, consequently, the refractive power of the eye as a whole. The closer the object viewed, the greater the degree of accommodation. When a person is reading small type corresponding to his visual acuity, there will be a point at which the letters begin to merge, and if the type is brought closer reading will become impossible. *The least distance at which the reading of small type is still possible at maximum accommodation is the nearest point of clear vision* (punctum proximum). The greater the distance between the object and the eye, the lower the degree of accommodation; finally, at a certain distance depending on the type of refraction accommodation will be at rest. The greatest distance at which the eye can see is called *the far point of clear vision* (punctum remotum). Its position depends on the type of refraction. In emmetropia this point lies at an infinite distance away because in the emmetropic eye parallel rays coming from infinity are brought to a focus on the retina (considering the small breadth of the pupil one can regard rays entering the eye from a distance of 5 m as parallel rays). In near-sightedness the far-point of clear vision is at a definite distance from the eye, which depends upon the degree of myopia. In hypermetropia this point lies in the imaginary negative space (beyond the eye), since, as has been noted, the hypermetropic eye is capable of refracting convergent light rays which are nonexistent in nature.

If the degree of myopia or hypermetropia is known it is easy to determine the position of the far-point of clear vision. For this purpose, 100(cm) should be divided by a figure indicating the degree of myopia or hypermetropia. For example, in 4.0 D myopia the far-point of clear vision lies at a distance of $\frac{100}{4}$ (cm)=25 cm.

The state of accommodation depends on its *length* and its *power* or *volume*. *The length* of accommodation is *the distance between the far point and the near-point* (punctum proximum) of clear vision. *The power* or *volume* of accommodation is determined by the number of diopters by which the eye is capable of increasing its refractive power during accommodation. The volume and length of accommodation depend on refraction: the smallest volume is present in near-sightedness, the greatest volume in long-sightedness, while emmetropia lies in between. The capacity for accommodation depends not only on refraction but also on the age of the particular person, since the crystalline lens becomes sclerosed and thickened with age and its elasticity decreases. A decrease in accommodation is indicated by the growing distance of the near point; practically it means that persons older than 40 need spectacles for near vision which should compensate for their diminished power of accommodation. This lowering of the power of accommodation with age is called *presbyopia* or *senile hypermetropia*.

Among the other changes in accommodation mention should be made of its weakening—*paresis* and *paralysis* as well as *increase* or *spasm*. *Paresis* and *paralysis of accommodation*, causing difficulty in or even loss of clear vision at a near distance are encountered after diphtheria, in certain cranial injuries, upon the injection of atropine into the conjunctival sac, in poisoning with methyl alcohol, plasmocide, antifreeze solution, in intoxication with *B. botulinus*, etc.

Spasm of accommodation may develop as a result of straining the eyes in work on near objects (particularly in children), or due to the administration of eserine or pilocarpine drops into the conjunctival sac. Spasm is manifested by the near-point of clear vision drawing closer to the eye and by the presence of temporary false near-sightedness.

CLINICAL PICTURE OF REFRACTION ANOMALIES. SELECTION OF SPECTACLES

Refraction anomalies give rise to certain clinical symptoms and complaints on the part of the sufferer. Since long-sighted persons have to exert their power of accommodation all the time they may develop signs of eye fatigue: sharp pain in the eyes, pain in the

orbital region and in the forehead, indistinct vision when reading small print, etc. This condition, known as *accommodative asthenopia*, is more common in high-degree hypermetropia. It may, however, also occur in persons with low-degree hypermetropia from intense work or study under unfavourable circumstances as well as at an age when concealed hypermetropia becomes obvious. Because of the continual exertion of accommodation and the convergence associated with it children with hypermetropic refraction may develop convergent strabismus.

Most people are hypermetropes (50-55 per cent), there are fewer emmetropes (30 per cent), and even fewer myopes (15-20 per cent). Almost every child is born a hypermetrope. The eyeball grows with the body and its anteroposterior axis elongates; hypermetropia diminishes and transforms into emmetropia or myopia. Under certain unfavourable conditions (inadequate lighting during work or study, overtiring the eyes with near work, incorrect design of desks for schoolchildren, irregular life, debility of children following infectious diseases, etc.) near-sightedness may grow worse. It comes on in junior schoolchildren and frequently becomes worse when they reach senior school age. Heredity also plays a certain role. The elimination of unfavourable conditions, measures to improve the child's general health, the prescription of spectacles for permanent wear and medical treatment are very important in the control of myopia. In view of this, before the beginning of the school year, the medical assistant or nurse in charge is obliged to examine the eyesight of all schoolchildren. Only the tenth row of letters in a chart for testing visual acuity is used for this purpose. Schoolchildren with visual acuity below 1.0 are registered on a special list and sent to an oculist for examination.

The subsequent clinico-prophylactic (dispensary) observation implies control over the wearing of correcting lenses, the medical treatment prescribed and the sanitary and hygienic measures taken. The latter include sufficient and uniform illumination in classrooms, seeing that schoolchildren with poor eyesight sit at the front desks, and that the other pupils sit at desks corresponding to their height. In an incorrect sitting posture the child may bend his head too low over a book, exercise book, etc., and cause eye strain which may lead to the development of myopia.

Children with progressive myopia should not take part in difficult sports such as weightlifting, boxing, wrestling, diving, springboard diving, etc., and are recommended to take up easier sports such as gymnastics, swimming, skiing; heavy physical exertion should be limited or forbidden. To avoid eye strain, such children should have a 10 to 15 minutes' rest every 45-50 minutes. The desk should be uniformly lighted by a lamp with a mat or green shade set at the left side. TV programmes lasting from one to two hours may

be watched two or three times a week at the most, wearing corrective spectacles and sitting at a distance of 2-3 m from the TV set.

In certain cases myopia grows worse to the point of malignancy, and is attended by opacity of the crystalline lens, so that the sufferer sees moving dots in his visual field. There are also pathological changes in the choroid and retina caused by the extension of the posterior pole of the sclera; this may lead to a large and even total loss of vision.

The most serious complication of progressive myopia is rupture and detachment of the retina which often result in a sudden, large and sometimes incurable loss of vision. The continual strain of convergence when viewing near objects causes weakness of the external ocular muscles known as *muscular asthenopia*; children of 6 to 10 years of age may develop divergent strabismus. Spectacles worn continually often help to prevent and sometimes to eliminate strabismus. Progressive myopia and possible complications may be treated and prevented by prescribing corrective spectacles for constant wear. Measures to improve the general health, tissue therapy (30 injections of aloe 3-4 times a year), vitamins A, B_2, D, E, etc., calcium chloride or its gluconate are all beneficial. Regular courses of such complex treatment are advised for high-degree myopia, particularly in complicated cases.

Sound sleep, fresh air, and light physical exercise are very important. Myopic children need special care after infectious diseases. They are suspended from school as long as possible since recovery from a disease is often followed by progressive myopia. In cases of markedly impaired vision (for example, in myopia complicated by changes in the internal tunics) ordinary spectacles are often of little help. In such cases it may be helpful to use *telescopic glasses*, which look like a pair of small binoculars set in a spectacles frame.

When there are refraction anomalies vision is corrected with optic lenses. A set of such lenses consists of spherical convex (+) and concave (—) lenses with powers of 0.25 to 20.0 D as well as cylindrical lenses; in addition, the set includes prismatic and colour lenses, spectacle frames and shutters. The power of each lens in diopters is indicated on the lens frame and near the lens nests in the carrying case.

In cases of hypermetropia convergent lenses (convex +) are used, which convert parallel rays into convergent rays; in myopia diffracting lenses (concave —) are used, converting parallel rays into divergent rays. When selecting a lens one has to determine the refractive power of the examined eye. It corresponds to the lens through which vision is best; this method is said to be *subjective* since the oculist is guided by the patient's answers.

The selection of lenses is begun with an examination of the patient's visual acuity. Then a 1.0 D convex lens and a 1.0 D concave lens are placed in front of each eye in turn, with the other eye covered. If vision improves with the convex lens this points to hypermetropia, and with the concave lens to myopia. The power of refraction is thus determined; lenses of increasing strengths are placed in front of the eye until the one through which vision is best is found. In hypermetropia one is prescribed the strongest lenses, and in myopia the weakest lenses of those through which vision is best. This is necessary for complete relaxation of accommodation in hypermetropes, which in this type of refraction is brought into play also in vision at a distance, and in myopes for preventing excessive strain in the act of accommodation.

In many cases, particularly in cases of slight and moderate refraction anomalies, the visual acuity may be raised to 1.0 with spectacles.

Failure to improve vision sufficiently with spherical lenses points to astigmatism, which may be corrected only with the aid of positive or negative cylindrical lenses. These lenses are sections of a cylinder and refract rays of light only in one direction. The other inactive optic meridian is the axis of the cylindrical lens. Such lenses, when used for simple astigmatism or in combination with spherical lenses for compound and mixed astigmatism, may improve vision considerably.

More accurate data is obtained by skiascopy, an objective method for determining refraction. It is carried out with the aid of an ophthalmoscope and skiascope (flat mirror).

The method of skiascopy is as follows. The eye is illuminated by an ophthalmoscope and the red reflex in the pupil watched as the ophthalmoscope is revolved first about its vertical axis and then about its horizontal axis. These movements cause a shadow to appear in the region of the pupil. This shadow may follow the movement of the ophthalmoscope or move in the opposite direction. In an examination performed through the concave mirror of an ophthalmoscope from a distance of 1 m the direction of shadow movement depends upon refraction. For example, in 1.0 D myopia no shadow movement is noted, in myopia of a higher degree the shadow moves to the same side as the ophthalmoscope, and in other types of refraction (myopia less than 1.0 D, hypermetropia, emmetropia) it moves in the opposite direction.

To ascertain the degree of refraction anomaly, skiascopy is continued by placing lenses of increasing strengths in front of the patient's eye until shadow movement is no longer noted. The last lens through which no shadow movement is noted will, upon correction by 1.0 D, indicate the degree of myopia or hypermetropia in diopters (in myopia one should add 1.0 D to the lens power, and

in hypermetropia deduct 1.0 D). This method can also be used to determine astigmatism by neutralizing the shadow (with lenses) in two mutually perpendicular meridians. The position of these meridians (axes) is determined by special instruments—ophthalmometers. Skiascopy is very important as an accurate and objective method for examining the power of refraction. It is especially valuable for determining a person's fitness for a particular job and for a medical examination of work capacity, etc.

For an exact determination of refraction anomalies in children and young persons the examination is performed with the pupil dilated by atropine (to have accommodation at rest).

When selecting spectacles for reading or close work for old people with presbyopia (senile diminished accommodation) one is guided by the relationship between the optic lens power and age. For this purpose, the visual acuity is first found for each eye and refraction is then determined with the aid of corrective lenses. People of 40 with emmetropic refraction are prescribed glasses of +1.0 D strength; those of 45 years +1.5 D; of 50 years +2.0 D; of 55 years +2.5 D; of 60 years +3.0 D; and those of 65 years and older +3.5 D.

In hypermetropes and myopes the above figures are taken as the basis but are corrected by the degree of refraction anomaly. For this purpose, in cases of hypermetropia the number of diopters corresponding to its degree is added to the lens strength corresponding to the patient's age. For example, a person of 50 with 1.0 D hypermetropia is prescribed a +3.0 D lens rather than a +2.0 D lens as is the case in emmetropia. And conversely, in myopia, in view of the excessive refractive power, the lens strength is lower, in accordance with the degree of myopia. A person of 50 with 1.0 D myopia is prescribed lenses of +1.0 D strength for near vision. If a patient wears spectacles all the time the lens strength is decreased for near vision in accordance with his age. For example, a person of 50 with 6.0 D myopia will use for reading concave lenses 2.0 D less in strength than those for distance, i.e., lenses of —4.0 D strength. The spectacles have been correctly selected if the patient can read print for near vision corresponding to his visual acuity.

The strength of any optic lens can be determined by the method of neutralization with the aid of a set of glasses. This method is based on the fact that when a positive lens is moved the object seen through it will appear to move in the opposite direction, and in the case of a negative lens in the same direction. When the type of lens has been determined it should be coupled with a lens of the opposite sign; the lens strength for which the object does not appear to move as the coupled lenses are moved will indicate the strength of the lens under examination. If, for example, movement of the object ceases when a +3.0 D lens is used the lens in question will have —3.0 D strength.

4

LAYOUT AND EQUIPMENT OF THE OPHTHALMOLOGIST'S OFFICE OF AN OUT-PATIENT CENTRE AND THE EYE DEPARTMENT OF A HOSPITAL

THE OPHTHALMOLOGIST'S OFFICE

The ophthalmological section should be housed in a light, spacious room at least 5.5 m in length (it will be recalled that examination of visual acuity is performed at a distance of 5 m). The chart for examining acuity of vision is set in a lighting device and hung on the wall in front of a window. The patient sits on a chair placed by the opposite wall, near a table with a set of optic lenses and type for reading. By the window or near another wall is a table, preferably surgical, covered with plate glass (Fig. 18). It is used for keeping bottles of eye drops and eye ointments which should be arranged in a definite order for convenience, and also eye baths (Fig. 19). Eye drops should preferably be kept in Stroschein's phials with ground-in pipettes (Fig. 19*c*) and should such phials be unavailable, in bottles for antibiotics with plugs with holes for pipettes. Eye drops should frequently be replaced with new ones and before use should be boiled in bottles or Stroschein's phials placed in boiling water, for example, in a sterilizer.* Liquids for irrigation and disinfection of the eye are best kept in undines (Fig. 19*a*). Silver nitrate, adrenalin (epinephrine), eserine, yellow mercury ointment, etc., should be stored in dark containers, since they are liable to decompose under the influence of light.

In addition to medicines, one should keep on the table a glass with sterile glass sticks for ointments, a trough of damp sterile cotton wool, a drum for dressing materials, a trough for sterile instruments—pincers, palpebral levators, needles for extracting foreign bodies from the cornea, etc.

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* For boiling the rubber tube is removed from a pipette and the latter turned upside down and inserted into the neck of the phial.

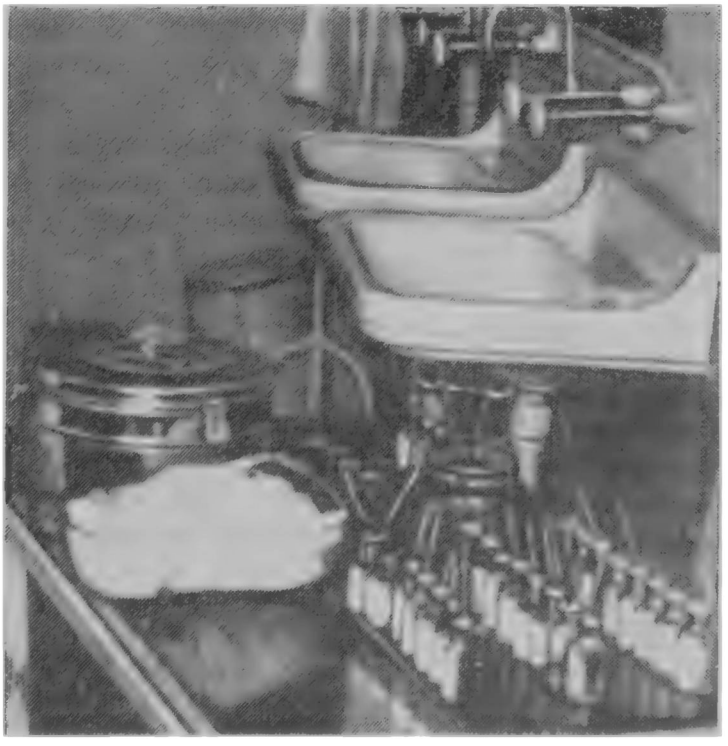


Fig. 18. A table for medicines and dressings in the dressing room of an eye clinic. Eye drops being administered by a nurse

Fig. 19.
a—undine; *b*—eye bath; *c*—eye drop Stroschein's phial with ground-in pipette

The table should be provided with two chairs set one against the other—one for the patient, and the other for the examiner (physician or doctor's assistant).

It is more convenient for the examiner to sit with the table on his right side. On the other table illuminated by daylight is placed a perimeter for examining the field of vision.

The office should be provided with a couch on which the patient is placed for taking measurements of the intra-ocular tension, for removing sutures, etc. There is a special cupboard for medicines and instruments.

The anterior section and fundus oculi are examined in a dark room with dark matt painted walls; the window is covered with black curtains. Instead of a separate room in the ophthalmological office one may put up a cubicle of plywood or dark dense fabric painted on the inside with black matt paint. Finally, the windows of a light room can be covered with black draw curtains. In a dark room there should be a table on which a lamp with a frosted electric bulb and an ophthalmoscope with two magnifying glasses are placed. Two chairs should be set near the table. The patient is seated at the table so that the lamp is behind and to the left and the examiner in front of him (see Fig. 24).

Before the examination is begun the nurse fills the bottles with fresh solutions of eye drops, sterilizes the instruments by boiling, prepares dressing materials, and sterile balls of cotton wool for wiping the margins of the eyelid after the application of drops and ointments. Dressings are prepared from whole bandages or strips

up to 1 m in length; it is wise to have strips of adhesive plaster 12 cm in length, with which the dressings can be secured to the head. The physician's table should be provided with forms for prescriptions and certificates and case histories of out-patients.

During a consultation the medical assistant or nurse may, on the doctor's instructions, examine the patient's visual acuity, field of vision, and colour vision. The data obtained are recorded in an out-patient's chart. The doctor's assistant should be capable of performing all the required manipulations: giving drops, applying ointments, taking smears from the conjunctiva for bacterioscopic examination, extracting superficial foreign bodies from the conjunctival sac and the cornea, expressing trachoma granulations, etc.

The ophthalmological room should be equipped with the following apparatus, instruments, medicines and medical utensils: (1) Roth's apparatus with charts for examining visual acuity in adults and children; (2) set of testing lenses; (3) ruler graduated in millimetres for measuring the distance between the pupils; (4) ophthalmoscope with two lenses (13.0 and 20.0 D); (5) table lamp with a frosted bulb for ophthalmoscopy (or oil lamp with round wick); (6) skiascopic rulers; (7) elastotonometer; (8) perimeter; (9) binocular lens; (10) Rabkin's charts for examination of colour vision; (11) needles and spuds for extracting foreign bodies from the cornea; (12) Desmarres' palpebral levators in two sizes; (13) dissecting, surgical and epilating ophthalmic forceps; (14) forceps for expressing trachoma granulations; (15) ophthalmic scissors—curved and straight; (16) ophthalmic scalpels; (17) needle-holder; (18) surgical ophthalmic needles (curved); (19) cone-shaped probes and Bowman's probes Nos. 1-4; (20) Weber's scalpel; (21) suturing silk; (22) syringes (of 2 g and 10 g); (23) Stroschein's phials for eye drops; (24) eye pipettes; (25) glasses for ointments; (26) ophthalmic glass sticks for application of ointments; (27) undines; (28) kidney-shaped enamelled basins; (29) small drums; (30) small sterilizer; (31) electric stove or primus-stove; (32) oxygen bag for introducing oxygen under the conjunctiva; (33) cardiac drugs.

LAYOUT AND EQUIPMENT OF THE EYE HOSPITAL

The eye hospital has a number of wards, a dressing room, an operating room with a room for preoperative treatment, a dark room, and auxiliary premises.

The layout and equipment of the wards are identical to those in other hospitals. In large hospitals there are special wards for patients with trachoma and purulent diseases of the eye. It is more convenient to have special wards for surgical patients. In these

wards, the bed-heads should be provided with special supports with which a patient can be put in a half-sitting position.

The dressing room should be well lit and spacious. The patient's chair is set facing a window; a dentist's chair is preferable for this purpose. The patient is seated so that his head lies on the headrest cushions. Nearby are two small tables, one with a selection of eye drops and ointments for treating trachoma and purulent diseases of the eye, and the other with similar remedies for the so-called clean patients. In addition, there should be a separate set of glasses with medicines for dressings applied to surgical patients. The tables with medicines, or preferably a separate small table, are used to keep the following materials and instruments: (1) a drum of sterile material—bandages, gauze patches with cotton-wool to be applied to the closed eye before bandaging; (2) a trough or pot with damp sterile cotton-wool balls for wiping the margins of the eyelid after the application of drops or ointments; (3) a trough for sterilized instruments: dressing forceps with which to take out dressing materials from a container, palpebral levators, pincers, probes for the lacrimal passages, needles for extracting foreign bodies from the cornea, and glass sticks for applying ointments.

Near a window is set a table for intravenous infusions, a table with an electric stove or an electric sterilizer. The couch on which the patient is placed for measuring the intra-ocular tension, for removal of sutures, etc., is set near a wall.

In the medicine cupboard there are special locked sections for toxic (A) and drastic (B) drugs. The first section contains solutions of atropine, scopolamine, pilocarpine, eserine, morphine, pantopon, cyanate, etc.; the second contains novocain, adrenalin, ether, etc. The other drugs are kept in the common section of the cupboard.

The dressing room should be provided with a portable electric lamp with a handle and long cord, which is used in bandaging and examining surgical patients in the dressing room and wards.

In a hospital without a physiotherapy room, the dressing room is provided with a table-mounted Sollux lamp, Minin's blue lamp with a reflector, and apparatus for electrophoresis.

Intermediate medical personnel daily prepare the dressing room for the admission of patients. White painted furniture and glass stands are wiped with cotton-wool soaked in mercuric chloride or oxycyanate and carefully wiped dry. Glass sticks and instruments are sterilized by boiling. Sterile cotton wool balls for wiping the eyelids are placed in a trough. Eye drop glasses and pipettes are first washed in a soda solution, and then boiled in distilled water, filled with the required solutions and again heated in a water bath. Solutions of antibiotics, sulphonamides, adrenalin, silver nitrate, collargol and riboflavin should not be boiled. Cocaine is

sterilized over three days for 20 minutes at a time at a temperature of 70°C. Glasses and phials for ointments should be provided with labels with the names of the medicines clearly written; labels are glued upon phials and glasses immediately after they have been filled.

Care should be taken that no pipette is taken by mistake from one solution and put into another. This is particularly important in the case of drugs for dilating the pupil (atropine, scopolamine, cocaine) or constricting it (pilocarpine, eserine, etc.). For example, if a patient with glaucoma is given a drug for constricting the pupil with a pipette for atropine, the pupil will dilate and this error may cause a dangerous attack of glaucoma. Even graver symptoms will be caused by a chance administration of as little as one drop of atropine into a glaucomatous eye. If pilocarpine or other drugs for constricting the pupil are mistakenly given instead of atropine the patient may suffer from inflammation of the uveal tract.

Therefore, the above drugs should be kept in definite places, at opposite ends of a table. It is wise to stain these drugs slightly, for example, pilocarpine with a drop of eosin, atropine with brilliant green. Bottles of atropine and scopolamine can be covered with a glass so that they are not mistaken for phials of pilocarpine and eserine.

In the morning, dressings are changed by the attending doctor aided by a medical assistant or nurse. First treated are the bedridden patients in the wards, where troughs with the required medicines specially prepared for the surgical patients, a sterilizing drum with dressing materials and an electrical lamp are brought for this purpose. The bandage is removed from the patient's eye by a medical nurse, and the gauze patch by the doctor, after which the doctor wipes the margins of the eyelid with moist tampons, and the nurse gives the eye drops. The doctor patches the eye with sterile gauze and the nurse applies the bandage and carefully shifts the patient or helps him take his seat as the doctor instructs.

The other patients are bandaged in the dressing room. Further manipulations during the day are performed by a medical assistant or nurse as prescribed by the doctor. Prescriptions are recorded either in the "prescription book" which is handed over to the shift next on duty, or on large sheets of paper, as well as in the case histories. Intravenous infusions, intramuscular injections, application of leeches to the temple, physiotherapeutic and other procedures are carried out after the dressings have been changed. Medicines to be taken per os are usually distributed before meals.

Intermediate medical personnel should see that the patients are placed in appropriate wards, should deliver samples (urine, blood, faeces, conjunctival smears, etc.) for analysis to the laboratory in

due time, should send patients to the x-ray room for examination, and to specialists for consultation, should measure the body temperature of patients, etc. The findings of analyses are written down in the case histories, and the temperature measurements are recorded on a special temperature chart.

It is also the duty of intermediate medical personnel to keep patients, wards and rooms with apparatus and medical equipment clean. The medical assistant or nurse should wipe complex optical instruments themselves rather than entrust this job to medical orderlies or cleaners.

Intermediate medical personnel work in shifts according to a rota and, in addition to carrying out the doctor's prescriptions and their nursing duties, also carry out health education work, read newspapers to the patients, see that the hospital regulations are observed by the patients and visitors, examine food parcels intended for patients, check upon the work of medical orderlies and cleaners, etc.

A medical assistant or nurse working at an eye hospital should bear in mind that most of the patients have poor eyesight or are totally blind. Intermediate medical personnel should therefore help patients to learn how to walk about the clinic and, whenever required, accompany them. Besides medical care, a bed-ridden patient with a binocular dressing needs to be helped to keep in touch with his family, read and write letters, and read books, newspapers, etc.

In the morning, when her shift is over, the nurse reports to the doctor about her round of duty and the condition of the patients.

The preoperative room adjoins the operating room and is part of the surgical department. It contains cupboards in which instruments, medicines and dressing materials are kept, a couch, a stretcher and a trolley or wheel-chair for bed-ridden patients. In some large clinics there are separate rooms for sterilization, the storage of sterilized materials and for preparation for sterilizing. Where there is no such room these operations are performed in the preoperative room. Some sterilizing drums are used to keep gowns, bed sheets, masks for personnel and patients, and others are intended for bandages, gauze and cotton-wool, gauze squares, cotton-wool wicks (long, tightly-rolled cotton-wool sticks with which to wipe wounds during an operation), wooden sticks with cotton-wool for painting the skin of the eyelid with brilliant green, and long, narrow gauze tampons for special operations.

Ophthalmic instruments are much smaller than conventional surgical instruments; they are very thin and frail and therefore require especially delicate handling. Cutting instruments are kept on a stand, and are tested for sharpness on a special device covered with very thin chamois leather. The instruments should always

be very well sharpened. They are sharpened in a special workshop, but an experienced surgical nurse may also do this job on an ordinary whetstone. There is a new special electrolytic apparatus which is very convenient for sharpening instruments.

The operating room has the following equipment: an operating table, tables, trolleys and stands for instruments and medicines, sterilizing drums and a sterilizer, a portable and a shadowless suspended lamp, a gaint electromagnet, a hand-operated permanent magnet, and apparatus for surgical diathermy. The surgical nurse prepares and sterilizes linen, dressing materials, suturing silk and catgut of the required gauge according to the rules of surgery: she is responsible for their sterility. Cutting instruments are immersed in alcohol not later than one hour before an operation; the other instruments are sterilized by boiling.

The medical nurse boils eye drops (atropine, pilocarpine, dicaine [tetracaine], etc.) in sterile bottles, and prepares fresh solutions of penicillin, streptomycin and albucid (it should be remembered that adrenalin and collargol solutions should not be boiled). For each operating day, fresh medicines are ordered from a chemist, and those remaining after an operation are handed over to the dressing room.

Before an operation, the surgical nurse treats her hands as before an operation and puts on a sterile gown and mask. Then she covers the table with a sterile bed-sheet and places on it the required instruments, pipettes, syringes, gauze, cotton-wool, bandages, solutions of novocain, dicaine, brilliant green, and a saline solution. The table is then covered with a sterile bed-sheet.

PREOPERATIVE TREATMENT. SURGERY AND POSTOPERATIVE CARE

Before an operation, particularly intra-ocular, the patient's cardiovascular system, respiratory passages and gastro-intestinal tract are examined; it is also necessary to make analyses of the blood, urine and faeces, measure the blood pressure, etc. It is exceedingly important to identify diseases likely to cause a post-operative purulent infection (purulent otitis, tonsillitis, furunculosis, pustular skin diseases, etc.). In such cases, as well as in a number of diseases of the cardiovascular system, the blood, lungs, etc., the operation is put off until the patient has recovered. An exception is made for emergency operations for penetrating wounds of the eye, acute glaucoma, serpiginous ulcer of the cornea, i.e., cases where the condition of the eye necessitates an operation before the prolonged general examination of the patient is completed. In emergency cases, antibiotics and sulphonamides should be used

extensively for prophylactic purposes. In these cases, however, the patient should also be thoroughly examined by the attending physician. It goes without saying that both eyes of the patient should, as a rule, be examined with care. The contraindications for an operation on the eyeball are as follows: suppurative inflammation of the lacrimal sac, styes and acute conjunctivitis. If the conjunctiva is apparently normal, an intra-ocular operation should be prefaced by irrigation of the lacrimal passages, and examination of a smear or a culture from the conjunctiva. If pathogenic organisms are detected (streptococci, pneumococci, staphylococci, Koch-Week's bacilli, etc.) the operation is postponed and appropriate medical treatment is undertaken.

Patients expecting surgery requiring incision of the eyeball are given drop instillations of penicillin or synthomycin solutions 4-5 times daily to disinfect the conjunctiva in the preoperative period. It is also desirable to administer 200,000-300,000 U of penicillin intramuscularly before an operation. On the eve of the operation, the patient is given an enema and a bath. The skin of the face and eyelids is washed with lukewarm water and soap, the conjunctival sac is irrigated with an antibiotic solution, and the skin between the ear and the middle of the nose is painted with a 1 per cent alcohol solution of brilliant green, and the eye protected with a monocular sterile bandage.

Before an operation, the patient is taken to the preoperative room where he is helped to take off his gown and footwear and to put on special cotton socks. A nurse plugs his ear on the side of the eye to be operated on with a piece of cotton-wool to prevent the entry of blood and makes three instillations of a 0.25-0.5 per cent dicaine solution into the eye. Then the patient is led into the operating room and placed on the operating table where the skin of the eyelid and face is painted with a thick layer of a 1 per cent alcohol solution of brilliant green, and the conjunctival sac is irrigated with penicillin solution.

The patient is covered with a bed sheet, and his face, head and chest are covered with a sterile cloth with an opening for one or both eyes.

This is followed by local novocain anaesthesia (subconjunctival, subcutaneous, intramuscular, retrobulbar). In adults, operations under general anaesthesia are performed in exceptional cases. Only restless children are sometimes given intramuscular or intravenous injections of pentothal or hexenal (hexobarbital) according to the rules of general surgery.

The scrub nurse should watch the progress of the operation and hand the surgeon the required instruments without delay and with strict observance of aseptic rules. The scrub nurse is also responsible for the sterility of surgical material.

The duties of the medical assistant or nurse in the operative care of the patient involve seeing that the doctor's instructions are carried out accurately. Intermediate medical personnel should see that patients are delivered to the operating room in due time according to the waiting list. It is also their duty to put the patient in a correct position on the operating table and keep an eye on his condition. They are responsible for his return to the ward on a stretcher or on foot. The nurse accompanies the patient as far as his bed and helps him to lie down. In accordance with the doctor's instructions, the nurse keeps watch on the patient's position in bed, his diet, evacuation of the bowels, and carries out the doctor's prescriptions. The patients should be given their meals in due time, washed and helped to keep their dressings in place.

Postoperative care of the patient is very important if the operation is to be successful. On the first day after an operation involving incision of the eyeball, the patient is given a small ration of exclusively liquid food because masticatory movements disturb the comfort of the eye. If too much food is taken before and especially after an operation it may cause vomiting so that the operative wound bursts open. After an operation for detachment of the retina, extraction of intra-ocular foreign bodies, or transplantation of the cornea, the patient should lie on his back for a long time. Special care should therefore be taken to make his bed comfortable for him. Special precautions are required when changing dressings and instilling eye drops so as to avoid separation of the lips of the wound and other complications. During the first 5-6 days, dressings are usually applied by a doctor.

5

EXAMINATION OF THE PATIENT

HISTORY TAKING

Examination of the patient is begun by finding out his complaints, which may point to a certain disease. For example, pain in the eye, excessive lacrimation and photophobia are usually observed in diseases of the anterior portion of the eyeball (cornea, iris and ciliary body) as well as of the eyelids and conjunctiva. Complaints of progressive loss of vision may be indicative of pathological changes in the crystalline lens, the choroid, the retina or the optic nerve, or of progressive myopia and simple glaucoma. A sudden loss of vision without painful sensations occurs in cases of acute obstruction of the retinal vessels (embolism, thrombosis, spasm), and in certain changes in the macula lutea and the optic nerve. Especially characteristic are patients' complaints during attacks of acute glaucoma when a sudden loss of vision is accompanied by a sharp pain in the eye and in the dependent half of the head, nausea and vomiting.

Later, one should collect detailed anamnestic data pertaining to the initial period of the disease: the time of onset, its duration and character (acute or chronic), the suspected cause, the treatment carried out and its efficacy; it should be found out whether there has been an injury to the eye, or contact with patients suffering from contagious diseases of the eye (trachoma, conjunctivitis, etc.).

It is highly important to establish the general condition of the patient, which systemic diseases he has had in the past or is suffering from at that time, since the cause of an eye disease may be rooted in various other diseases. For example, inflammation of the palpebral margins and styes may be associated with anaemia, scrofulosis, disturbed functioning of the digestive tract, metabolic disorders, and helminthiasis. Lesions of the choroid and retina

and of the optic nerve may be caused by infectious diseases, diseases of the paranasal sinuses, vascular hypertension, diabetes, diseases of the kidneys, of the blood, toxaemia of pregnancy, and diseases of the central nervous system.

When questioning the patient, it is necessary to find out his living and working conditions which may also be responsible for his illness or contribute to its manifestations. For example, chronic conjunctivitis is often associated with work in dusty surroundings (flour mills, tobacco factories) or with inadequate protection of the eyes against bright light (in electric welding, steel smelting, metal rolling). An increase in the intra-ocular tension and an attack of glaucoma may be provoked by physical fatigue, a sharp change in the ambient temperature, and nervous shocks.

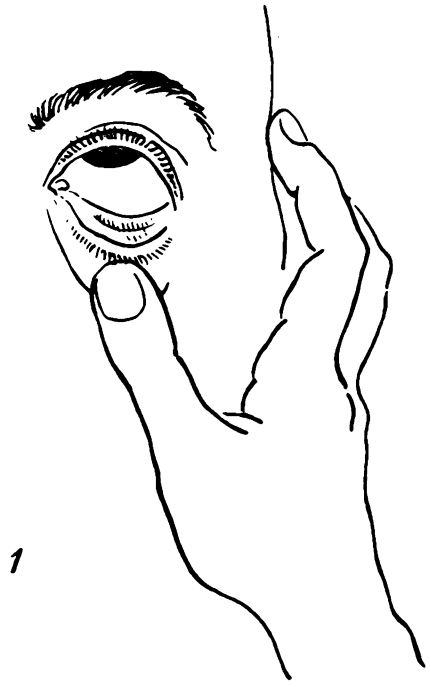
The examination procedure includes analysis of the blood, urine and faeces, x-raying, measurement of blood pressure, etc. Whenever required, other specialists may be invited for consultation.

Examination of the eye and surrounding parts should be carried out according to a plan and in a definite anatomical sequence so as not to overlook individual symptoms necessary for identifying the disease. Both eyes must be examined, beginning with the normal one.

EXAMINATION OF THE EYELIDS, CONJUNCTIVA, CORNEA, AND IRIS

The examination of the patient is begun with *an inspection of the eyelids*; the condition of their skin and margins, their position (inversion, eversion), the growth of the eyelashes, the width of the palpebral fissure, the presence or absence of photophobia, lacrimation and spasm of the eyelids are established.

Examination of the conjunctiva is possible only when the eyelids are everted. The lower eyelid is easily everted: for this purpose it is drawn downwards and pressed slightly against the bony edge of the orbit, the patient looking upwards (Fig. 20,*1*). This brings into view the mucosa of the lower eyelid and the palpebral fold. To examine the mucosa of the upper eyelid the patient should be told to look downwards. The examiner puts the palm of his left hand on the patient's forehead and draws the upper eyelid upwards with his thumb, while with the thumb and forefinger of his right hand he takes hold of the palpebral margin and draws it downwards (Fig. 20, *2*). This brings into relief the cartilage under the skin; at its upper margin the eyelid is pressed with the thumb of the left hand or with a glass stick and is everted as though on a fulcrum (Fig. 20, *3*). After the eyelid has been everted the finger or glass stick should be removed and the everted eyelid pressed against the eyeball. To examine the upper palpebral fold the eyeball is



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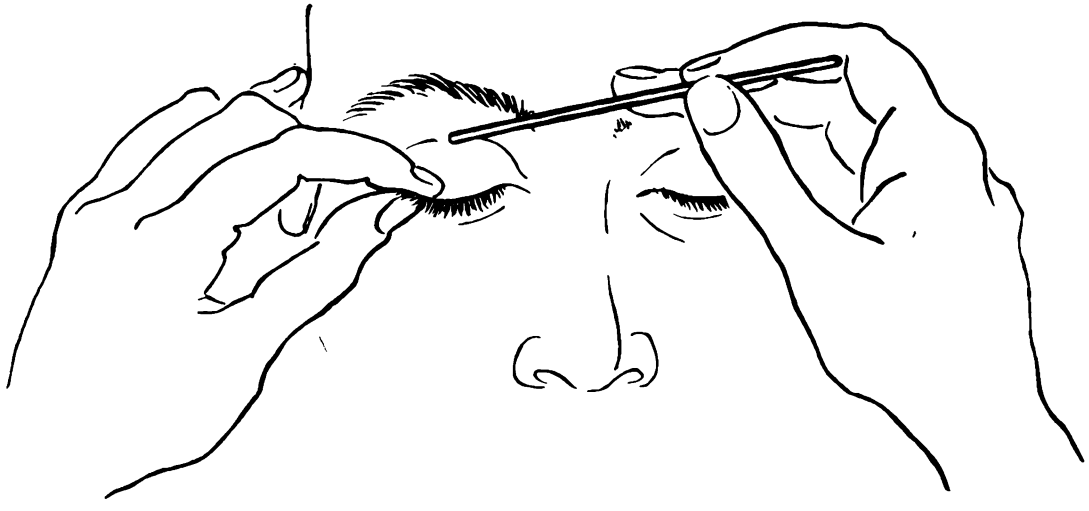


2

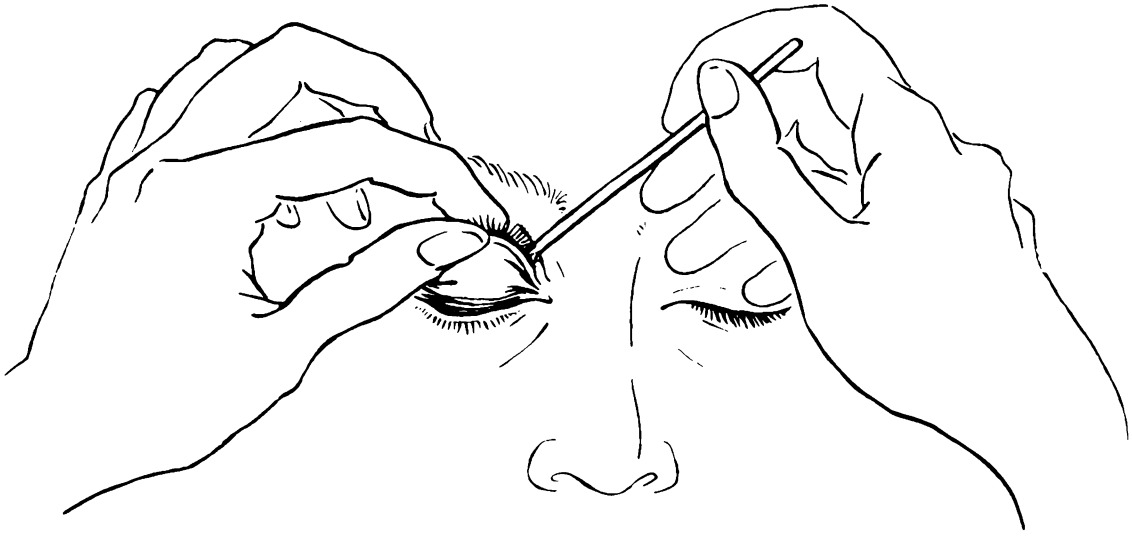
Fig. 20. (1-4) Examination of the conjunctiva

1—eversion of the lower eyelid;

2—eversion of the upper eyelid—first procedure

3—eversion of the upper eyelid—second procedure

4—eversion of the eyelid for examination of the superior palpebral fold



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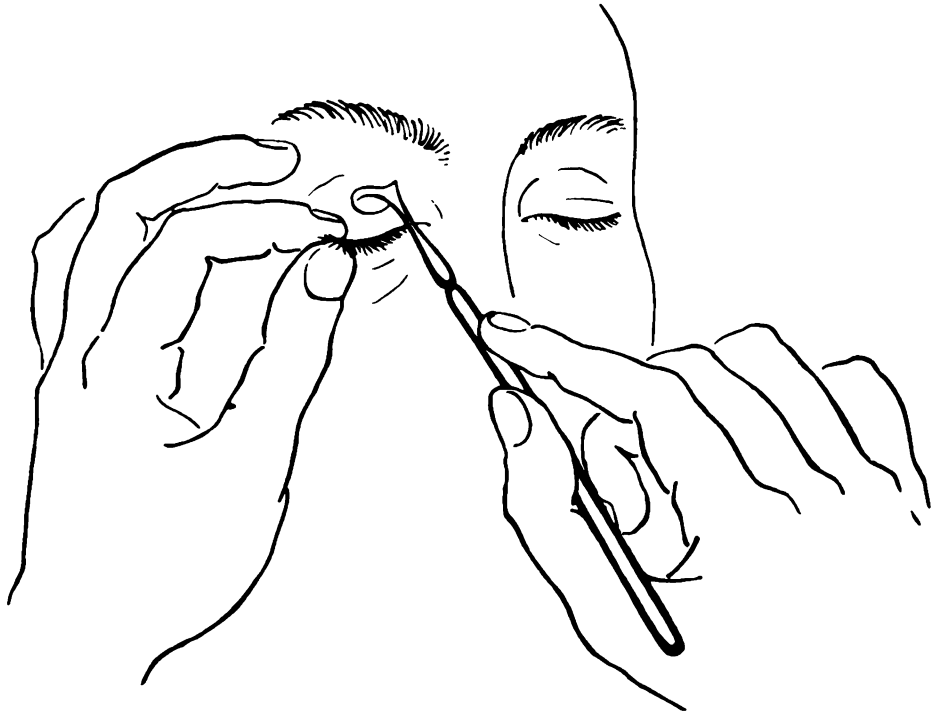

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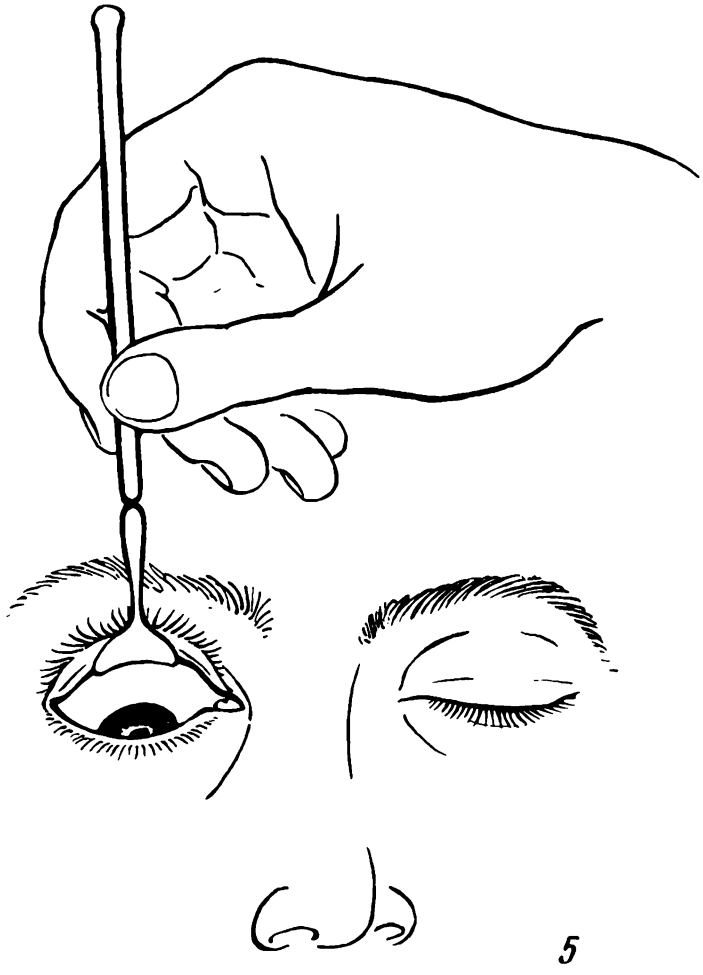


Fig. 20. (5) Examination of the conjunctiva
5—eversion of the upper eyelid by means of a palpebral levator for examination of the palpebral fold

pressed with the right hand through the lower eyelid and shifted upwards under the upper eyelid. The latter is drawn upwards with the left hand and pressed against the orbital margin (Fig. 20, *4*).

If such procedures fail to evert the upper palpebral fold sufficiently, one can use a palpebral levator which is placed with its convex end on the palpebral skin above the cartilage and used as a lever to evert the eyelid, drawing it with the end of the instrument and supporting, if necessary, the ciliary margin of the eyelid with the finger at the upper edge of the orbit (Fig. 20, *5*).

All manipulations should be carried out with caution, particularly in cases of ulcers of the cornea, or injuries to the eye, since inadequate care may result in perforation of the cornea. To avoid such a risk, the patient should be told to sit still and not to move his eye and head.

If there is copious secretion from the conjunctiva, the margins of the eyelid should first be wiped with a moist tampon and then

slowly separated; the conjunctival sac is washed out with an antiseptic solution and the eyelids then everted. This is necessary to prevent pus from getting into the examiner's eyes. It is also wise for the examiner to wear shielding glasses.

During examination of the conjunctiva one should note its colour (hyperaemia or anaemia of the mucous membrane), its thickness and find out whether or not it has follicles, scars, false membranes and secretion. If the latter is found, a sample should be taken for bacterioscopic investigation. The secretion is taken from the conjunctival sac with a thin slide or platinum loop, spread in a fine layer on a clean glass slide, dried in air, fixed by a single exposure to the flame of a burner and then stained with methylene blue or after Gram. For a bacterioscopic examination, a sample of the secretion is inoculated in a culture medium.

When examining *the lacrimal passages*, attention is given to the condition of the skin in this region and the position of the lacrimal puncta; it is also determined whether there is stagnation of tears overflowing the rivus lacrimalis, lacrimation, purulent or mucous discharge from the lacrimal puncta when pressure is applied to the lacrimal sac, and whether the lacrimal passages are clear.

To examine the anterior portion of the eye in children, especially in spasm of the eyelids, the palpebral fissure is opened with palpebral levators (Fig. 21). Examination of the eye is usually carried out in a dark room with the aid of *lateral* or *focal* illumination. For this purpose a source of light is set to the left and in front of the patient on a level with his head, and the rays of light from the lamp are brought to a focus on the cornea with a 13.0-20.0 D biconvex lens. Such illumination brings into view small changes in the cornea, the iris, and the anterior portion of the crystalline lens. They are even more visible with lateral illumination through a second lens of 13.0 D strength set in front of the eye at its focal distance (Fig. 22) or through a binocular lens attached to the examiner's head.

The most convenient equipment for examining the anterior portion of the eye is a slit-type lamp coupled with a corneal microscope.

During *an examination of the cornea*, attention is given to its size, form, transparency, lustre and reflecting power. These properties are altered in an inflamed cornea and it may be pierced with blood vessels. Sometimes its rear surface is found to have whitish or brown dots (precipitates) due to deposits of pigment and exudate which form in inflammatory diseases of the uveal tract.

The sensitivity of the cornea is determined by touching it with cotton-wool fibres; this normally causes closure of the eyelids (corneal reflex) and a sensation of touch.

Fig. 21. Examination of the eye with the palpebral fissure kept open by palpebral levators

Fig. 22. Examination in a lateral or focal light with the aid of two loupes

Using focal illumination, one can adequately examine *the anterior chamber* and its contents—*the aqueous humor*. Normally it is transparent but in diseases of the eye it may change due to the presence of blood (hyphaema) or pus (hypopion). One can clearly see *the iris* which normally has a clear-cut, distinct pattern. In inflammatory conditions a pale iris takes on a dirty-green colour, and a dark iris a rusty colour; its pattern becomes blurred and indistinct, and the pupil acquires an irregular shape owing to the formation of adhesions with the crystalline lens (posterior synechiae) or with the cornea (anterior synechiae). When the crystalline lens becomes less transparent the region of the pupil becomes grey in colour, and when there is pus in the vitreous body the pupil appears to be green.

When the iris is examined *the reaction of the pupil to light, accommodation and convergence are also studied*. Each eye is examined separately. With one eye covered the other eye is illuminated and the constriction of its pupil noted (direct reaction); then with one eye illuminated the reaction of the second pupil is examined (sympathetic). The patient is told to look into the distance and then at a near object (reaction to accommodation and convergence). In all such examinations a normal pupil quickly narrows.

OPHTHALMOSCOPY
(EXAMINATION WITH AN OPHTHALMOSCOPE)

The remaining parts of the eyeball (*crystalline lens, vitreous body, ocular fundus*) are visible when examined with an ophthalmoscope, a concave mirror with a small central aperture (Fig. 23).

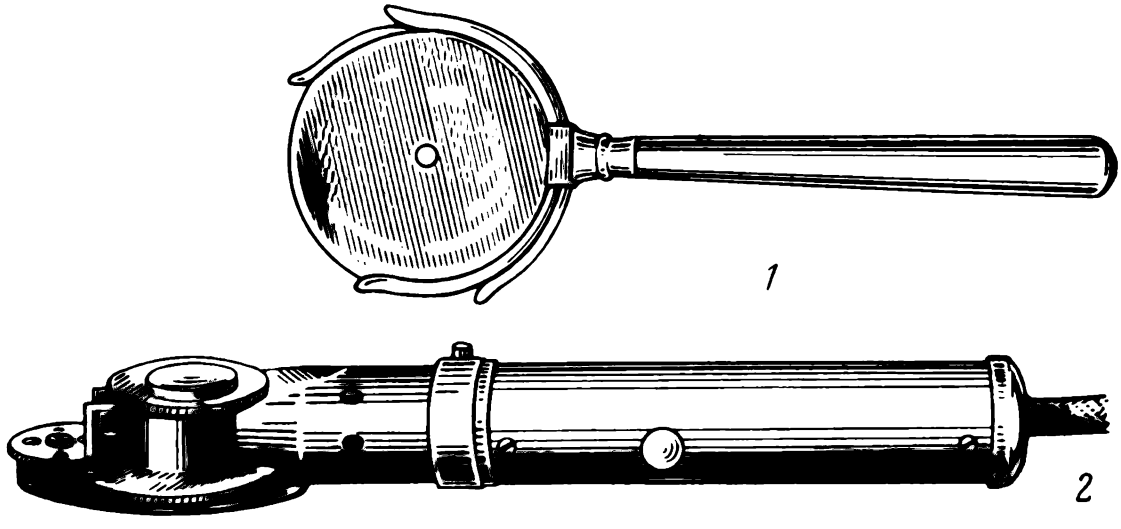


Fig. 23. Ophthalmoscope
1—mirror-type; *2*—electrical

Fig. 24. Examination with a mirror-type ophthalmoscope (lamp set to left of and behind patient's head)

This instrument was invented by Helmholtz in 1850. Ophthalmoscopy is a highly important diagnostic procedure not only in diseases of the eye but also in many diseases of the visceral organs and the central nervous system, since it enables the examiner to see the eye-ground, the optic disc, the retina and its vessels, and the choroid. The fact that the optic nerve is part of the brain extending to the periphery explains why the condition of the optic disc is affected by many diseases of the central nervous system, inflammatory and degenerative processes, and tumours. The vessels of the choroid are a continuation of those of the brain. They readily respond to systemic diseases, and are especially frequently affected by vascular hypertension, diseases of the blood and kidneys, diabetes, infectious and many other diseases.

To examine *the ocular media of the eyeball, the method of examination in transmitted light is used*. The patient is seated in a dark room, and the lamp is set behind and to the left of him on a level with his head. The eye is illuminated by light reflected from the

ophthalmoscope, so that the red reflex in the pupil can be seen as a result of rays of light being thrown back from the eye-ground. If there are any opaque parts in the path of the light rays, dark spots, strips, etc., will be seen against the background of the red reflex. When the eye is rotated sideways these opaque parts move towards the corresponding side if they lie in front of the centre of rotation of the eye (in the cornea or the anterior portions of the crystalline lens), or in the reverse direction if they lie behind the centre of rotation of the eye (posterior pole of the crystalline lens, the vitreous body).

In examination of *the ocular fundus—ophthalmoscopy*—the eye is illuminated by light reflected from the ophthalmoscope, and a 13.0 D lens is held at a distance of 7.5 cm (i.e., its focal length) in front of the patient's eye (Fig. 24). The rays of light entering the eye are reflected from the eye-ground, pass through the lens and converge in front of it, at its focus. This enables the examiner to see an inverted, magnified and real image of the patient's fundus oculi.

For the examiner to be able to see the optic disc (see Fig. 7), which lies inwardly to the posterior pole of the eye, the patient is told to look at the examiner's ear on the side in question. When the patient is looking straight into the ophthalmoscope, the region of the macula lutea is brought into view; it is normally shaped like a horizontal oval rimmed with a brilliant light band. When the eye-ground is examined, attention is paid to the colour and borders of the optic disc, the state of the tissue and vessels of the retina and the choroid, and whether they contain any inflammatory foci which may be encountered in diseases of the kidneys, cardiovascular and central nervous systems, in tuberculosis, syphilis, diabetes, and other diseases.

DETERMINATION OF THE INTRA-OCULAR TENSION

The intra-ocular tension which, as has been noted above, depends on the blood pressure in the capillaries and on the pressure of the intra-ocular humor against the walls of the eyeball, is very important in the examination of the organ of vision. It normally ranges from 18 to 28 mm Hg. Any increase or decrease has an adverse effect on the functioning of the eye as a result of disturbed ocular nutrition. An increase in the intra-ocular tension is the principal sign of glaucoma, a grave eye disease.

The state of the intra-ocular tension must be taken into account in treating patients, prescribing medicines and determining whether surgical interference is necessary.

The intra-ocular tension is often estimated by means of palpation with the fingers so as to compare the tensions in a sick and normal eye. The patient is told to close his eyes and look downwards, while

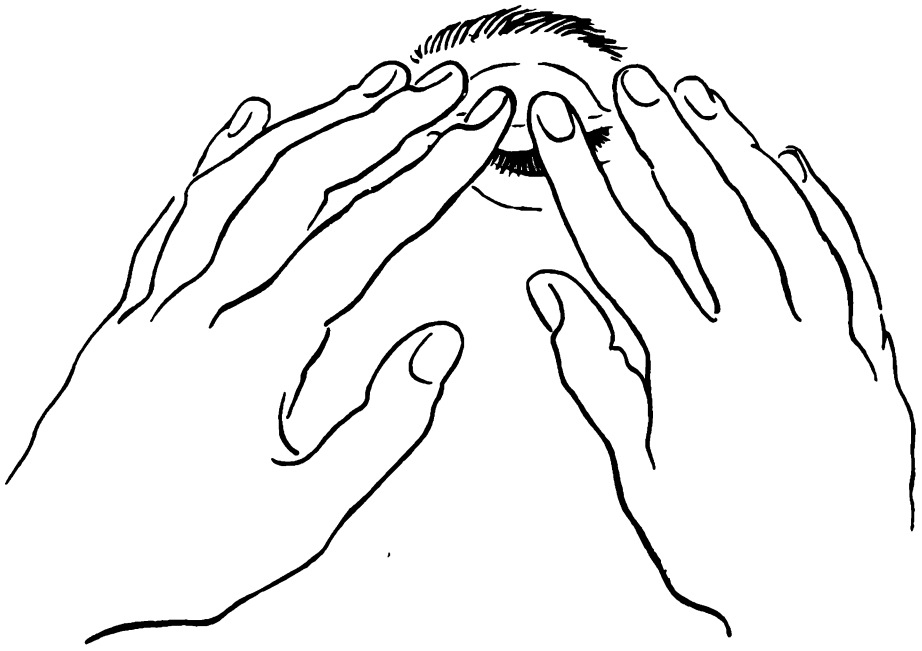


Fig. 25. Examination of intra-ocular tension by finger palpation

the examiner places his forefingers upon the upper eyelid above the cartilage and applies slight pressure to the eye with each finger alternately (Fig. 25). The degree of tension in the sick eye is estimated by comparing the tones (resistance) in the normal and the sick eye. The degree to which the tension has increased is recorded by one (+), two (++) or three (+++) symbols and the degree to which the tension has decreased by the respective minus symbols.

This examination gives only a tentative idea of the degree of intra-ocular tension, yet it has practical significance since it is not always possible or permissible to use the instrumental method (for example, in ulcers of the cornea, penetrating wounds, and after surgical operations).

An accurate estimation of the intra-ocular tension is usually made by means of special instruments called tonometers. The most precise of these is Maklakov's tonometer (Fig. 26*a*), which is a metal cylinder 10 g in weight, with flat ends made of porcelain which before examination are painted with stain made from collargol according to the following recipe:

Rp. Collargoli 2.0
Aq. destill.
Glycerini āā gtt. XXX

The stain is kept in a glass bottle with a ground-in stopper.

The stain, a 0.25 per cent dicaine solution, a bottle of alcohol, wooden sticks tightly wrapped in cotton-wool, and moist sterile

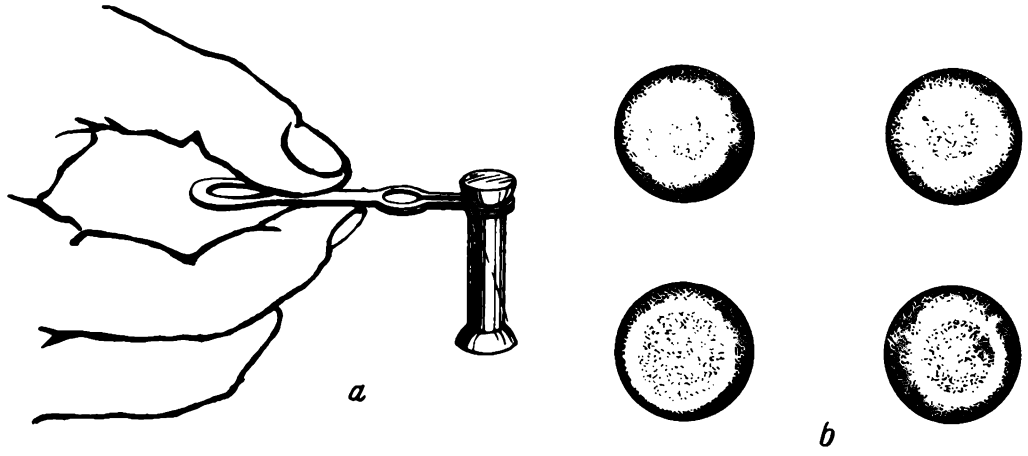


Fig. 26. Maklakov's tonometer (a) and prints (b) produced after measurement of the intra-ocular tension

cotton-wool are placed in a special trough. The patient is told to lie down on his back, the eye is anaesthetized with a 0.25 per cent dicaine solution, and the tonometer which moves freely on a special handle is applied to the cornea. In the place where the tonometer and cornea come into contact the stain is washed off to leave a pale circle (Fig. 26*b*) which is impressed on a sheet of paper soaked in alcohol. A special scale supplied with the tonometer set is used to measure the diameter of the circle, which corresponds to a certain degree of tension expressed in mm Hg. The smaller the pale circle, the higher the tension, and vice versa.

An even more accurate measurement of the intra-ocular tension is made by means of elastotonometry (after V. P. Filatov and S. F. Kalf's method) whereby the intra-ocular tension is measured consecutively with four tonometers of different weights (5, 7.5, 10 and 15 g). The findings are plotted in the form of a so-called elastotonometric curve. Electron tonometers and tonographs have recently been introduced into clinical practice.

An examination of the eye is rounded off by determining its position in the orbit and its motility. If an injury to the orbital walls or a foreign body inside the eye or orbit is suspected, x-ray photography of the eyeball and the orbit is required.

Examination of the eye should be undertaken after a study of the functional state of central and peripheral vision (see Chapter III) since the latter may change under the action of bright light during ophthalmoscopy and lateral illumination, after palpation, eversion of the eyelids and other manipulations.

6

METHODS OF TREATING EYE DISEASES

The organ of vision is very often involved in systemic diseases, therefore, after the diagnosis has been made and the cause of the disease determined, remedies affecting the body as a whole, i.e., the principal disease, should be used whenever required. In addition to this, measures are taken to regulate the diet, regimen, working and living conditions, and to eliminate occupational hazards, etc. Finally, drugs applied locally, directly to the eye, are very important in the treatment of eye diseases.

GENERAL THERAPY

The following are of special significance in the therapy of eye diseases: sulpha drugs, antibiotics, vitamins, physiotherapy, tissue preparations, and roborants.

Sulphonamides and *antibiotics* are used in the treatment of suppurations (acute conjunctivitis, gonoblennorrhea, stye, abscesses and phlegmons of the eyelids and the lacrimal sac, and purulent ulcers of the cornea), penetrating wounds, inflammatory diseases of the uveal tract, thrombosis of the orbital veins and the cavernous sinus, etc. Of the sulpha drugs norsulphazol (sulphathiazole), sulphadimezine, ethasole and other medicines are used. Ethasole is very effective for trachoma and other virus infections. It should be remembered, however, that in addition to general toxic symptoms these drugs may cause accommodation spasm simulating myopia, which goes away when the particular drug is withdrawn. The combined administration of sulphonamides and antibiotics produces excellent therapeutic effects.

Penicillin is used for inflammatory conditions associated with brucellosis and syphilis, for diseases of the optic nerve apparatus

(neuritis, opticochiasmal arachnoiditis, etc.), for gonoblennorrhea, phlegmons of the eyelids, lacrimal sac and orbit, for thrombosis of the orbital veins and the cavernous sinus, and for penetrating wounds of the eye.

Streptomycin is most widely used for the treatment of tuberculous diseases of the eye. It is indicated in the same cases as penicillin, and is particularly effective in combination with penicillin.

Synthomycin, *levomycetin* (*chloramphenicol*), *biomycin*, and *terramycin* are administered per os for trachoma, gonoblennorrhea, virus infections, ulcers of the cornea and other diseases.

It should be remembered that antibiotic therapy sometimes causes toxic symptoms in patients (skin rashes, dyspepsia, fungal diseases, etc.) and local manifestations of individual intolerance (oedema of the eyelids and mucous membranes, itching, hyperaemia of the skin and conjunctivitis). In such cases this treatment should be ended.

Phthivazide and *PAS* for peroral administration are prescribed for tuberculous and tuberculous-allergic diseases of the eye.

The activity of antimicrobial drugs is increased by simultaneously administering vitamins, desensitizing drugs, blood transfusions, etc.

Vitamin therapy is one of the most important remedies in ophthalmology, the vitamins usually being administered in combination. The widest use is made of the vitamins A, B_1, B_2, B_{12}, C, E, P, PP, etc.

Vitamin A in cod-liver oil and in dragee form is prescribed per os for scrofulosis, tuberculosis, blepharitis, relapsing stye, nyctalopia, pigmentary degeneration of the retina, and atrophy of the optic nerve. Vitamin A is present in carrots, tomatoes, milk, blackcurrants, butter, eggs, and liver. Therefore these foodstuffs are recommended for the diet of patients with the above-mentioned diseases.

Vitamin B_1, *thiamine*, is used in powder form or is injected intramuscularly in the form of a 5 per cent solution in doses of 1 ml every other day or intravenously in the same dose, usually in combination with 10-20 ml of a 40 per cent glucose solution. This vitamin is given for diseases of the optic nerve apparatus: neuritis, atrophy of the optic nerve, pigmentary degeneration of the retina, glaucoma, opticochiasmal arachnoiditis, and for certain diseases of the cornea (keratitis).

Vitamin B_2, *riboflavin*, is prescribed in powder form in a dose of 0.005-0.01 g per administration in combination with vitamin C in a dose of 0.05-0.1 g (and larger, when indicated) for relapsing stye, chronic blepharitis, eczema of the palpebral and facial skin, chronic conjunctivitis, diseases of the optic nerve apparatus, primary cataract, keratitis, ulcers of the cornea, and other diseases.

Vitamin C, ascorbic acid, is present in fresh fruit and vegetables (tomatoes, onions, garlic, lemons, black-currants, rose hips, oranges, etc.). Vitamin C is indicated for haemorrhage in the retina and the vitreous body, ulcers of the cornea, burns, infectious, toxic and allergic diseases, lesions of the optic nerve apparatus, and the initial stages of senile cataract, etc.

Vitamin B_{12} is injected intramuscularly in doses of 1 ml twice a week for anaemia, haemorrhages in the retina, and diseases of the optic nerve.

Vitamin D should be prescribed with calcium chloride for scrofulous and tuberculous diseases of the eye. The best effect is produced by vitamins acting in combination; it is therefore wise to prescribe several vitamins at once.

Tissue therapy, suggested by V. P. Filatov, consists in using for therapeutic purposes preserved tissues of animal or plant origin. These preparations, acting on the entire body, intensify metabolism, improve physiological bodily functions, increase body resistance, and contribute to healing and resorption processes. Tissue preparations—extracts of aloe, placenta, etc., are injected intramuscularly or subcutaneously in daily doses of 0.2-1 ml. A course of treatment of 45-50 injections is repeated 3-4 times a year. In addition to injections, the grafting of preserved tissues—placenta, aloe leaves, etc.—is undertaken. Such treatment is indicated for glaucoma, diseases of the optic nerve apparatus, keratitis, complicated myopia and eye injuries, in combination with other methods of treatment for a given disease.

Calcium chloride is prescribed as a desensitizing agent for allergic diseases (scrofulosis, spring catarrh, eczema, etc.), tuberculous diseases, intra-ocular haemorrhages, etc. Calcium chloride is used as a 10 per cent solution for intravenous injections, as a 5-10 per cent solution per os and as a 0.25 per cent solution for subcutaneous or intramuscular injections. In this case the initial dose is 0.2 ml which is increased each day by 0.2 ml to the final dose of 2 ml. During the next ten days the preparation is administered daily in a 2 ml dose. For prolonged treatment, it is wise to prescribe calcium *gluconate* in tablets of 0.5-1 g to be taken per os three times daily during a period of from six weeks to two months.

Dimedrol (diphenhydramine) is also used as a desensitizing agent and, in addition to that, as an antihistamine remedy for alleviating pain in glaucoma, and acute inflammatory diseases of the cornea and the uveal tract. It is prescribed in a 0.03-0.05 g dose to be taken three times daily over a period of 5-15 days.

Glucose in a 40 per cent solution is injected intravenously in a dose of 10-20 ml each day or every other day. The total dosage per course of treatment is from 15 to 20 injections. It is usually prescribed in combination with ascorbic acid or thiamine for in-

flammatory diseases of the cornea, the optic nerve apparatus, glaucoma, etc.

Diamox or *diacarb* analogous to it, *phonurite* and *hypothiaside* are used for reducing the intra-ocular tension, and are administered per os in a dose of 0.15-0.25 g from two to four times a day in combination with mioties applied locally (hypothiaside two times at most). The drug can be used for two weeks according to the doctor's prescription. The effect of diamox ends when it is withdrawn.

Another widely used method for treating eye diseases is *resorption treatment*, which is indicated in combination with other methods in cases of opacity and haemorrhage in the vitreous body, in inflammatory diseases of the uveal tract, the retina and the optic nerve, and for penetrating wounds, etc. A 10 per cent solution of sodium chloride or sodium iodide is injected intravenously in a 10 ml dose; good results are obtained from autohaemotherapy (5 ml of blood by intramuscular injection on alternate days) as well as from repeated blood transfusions in doses of 100-150 ml at intervals of 10-14 days.

Revulsive therapy—saline purgatives, round cupping glasses, foot baths, leeches—is prescribed in combination with other remedies and methods of treatment for acute inflammatory conditions in the uveal tract and for acute glaucoma. Leeches are applied to the temple; if they are unavailable the temporal skin may be incised in several places and from 10 to 15 ml of blood abstracted by means of a wet or common cupping-glass or Heurteloup's artificial leech (hollow cylinder with tight piston). Preference should be given to live leeches *which draw blood and simultaneously increase its clotting time.*

Specific therapy is prescribed for syphilis, tuberculosis, malaria, relapsing fever, diphtheria and many other diseases attended by pathological changes in the eyes.

Great attention is paid to the condition of the central nervous system and the appropriate therapy.

When there are trophic disorders of the eye tissues, particularly of the cornea (ulcers, neurotrophic keratitis) and pains caused by diseases of the uveal tract, glaucoma, etc., various forms of *novocain blocking* are used. For example, novocain is injected intravenously (a 5 ml dose of a 0.5-1 per cent solution) along the course of the orbicular muscle of the eye (a 5 ml dose of a 1 per cent solution), behind the eyeball (retrobulbarly), or under the conjunctiva of the eyeball in a 2 ml dose of a 1 per cent solution.

General invigorating therapy plays an important role in the treatment of eye diseases. Ferrum and arsenic preparations are used for this purpose as well as dietotherapy, climatotherapy, etc. There are often diseases of the oral cavity and the paranasal sinuses to be cured, helminths to be eliminated, etc.

PHYSIOTHERAPY, X-RAY AND RADIOTHERAPY

The entire complex of physiotherapeutic remedies, such as thermotherapy, ionization, ultraviolet light and UHF therapy, paraffin therapy, massage, diathermy, etc., is used in the treatment of eye diseases.

Thermotherapy—sollux lamps, hot-water bottles, warm compresses, paraffin applications—is used to dilate the superficial and deep-lying vessels, in order to increase resorption of inflammatory infiltrates. This treatment is prescribed for inflammatory conditions in the appendages and the anterior portion of the eye, particularly those causing pain.

Electrophoresis helps drugs to penetrate quickly deep into the tissues. Among the drugs applied are calcium chloride, potassium iodide, atropine, dionine, novocain, antibiotics and other preparations.

Ultraviolet light therapy with light from a quartz lamp is prescribed for scrofulosis and tuberculosis. It is contraindicated for a febrile condition, active tuberculosis of the visceral organs and glands, and for anaemia. Before treatment it is therefore necessary to consult an internist or a pediatrist. During total irradiation from a quartz lamp the eyes should be protected against direct ultraviolet rays.

Transthermia with ultrahigh frequency currents is prescribed to warm the deep tissues of the eye. It is indicated for acute suppurative inflammations of the eyelids, the lacrimal organs and the cornea. Irradiation is carried out daily or every other day with exposures of from 4 to 15 minutes, with a total of 10 to 15 sessions per course of treatment. This therapy is contraindicated for febrile conditions, fresh haemorrhages in the eye, malignant tumours, and vascular hypertension.

X-ray and radiotherapy is prescribed for malignant tumours of the eyelids, as well as after the removal of the eye and orbital contents for such newgrowths. X-ray therapy in small dosages is helpful in many diseases of the eye, such as inflammations of the uveal tract, absolute glaucoma, penetrating wounds of the eye, etc.

X-ray therapy of the eye should be carried out only under the direction of an experienced specialist.

LOCAL THERAPY

The exposed position of the eye makes it possible to use various medicines through irrigation, drop instillation, and application of powders and ointments. Solutions are introduced into the conjunctival sac, subconjunctivally, retrobulbarly and by injection and electrophoresis.

Fig. 27. Drop instillation

Irrigation of the conjunctival sac with a stream of liquid is undertaken in cases of eye burns, for superficial foreign bodies, secretion, and for disinfection of the conjunctiva before an operation on the eyeball. Among the agents used are physiological salt solution, antiseptic solutions of cyanide or oxycyanide of mercury, boric acid, potassium permanganate, and soda. Irrigations are carried

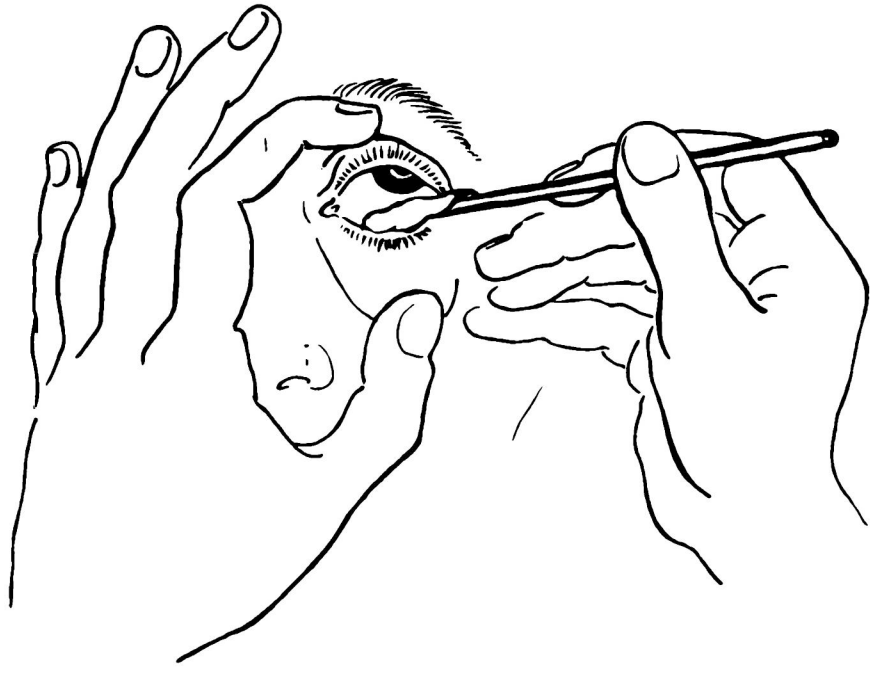


Fig. 28. Application of ointment

Fig. 29. Application of powdered medicine to the conjunctiva on a glass stick

out with the aid of an undine, a rubber bulb or an eye-bath, the latter being applied to the eye. In an emergency the eye can be irrigated simply by wringing out above it a piece of cotton-wool soaked in one of the above solutions. Before irrigation the eyelids are separated or everted (whenever required they are closed or opened). A trough or a kidney-shaped basin is held under the patient's chin to collect used liquid.

Eye drops are used most frequently; they are instilled in the following way. The patient is told to look upwards (Fig. 27), and the lower eyelid is drawn down with a wet piece of cotton-wool held in the left hand. The pipette is held at an angle to the eye with the index and middle fingers and 1-2 drops of medicine are poured into the lower fornix conjunctiva, after which the margins of the eyelid are wiped with moist cotton-wool. To avoid contamination of the pipette care must be taken that its end does not touch the eye or the instilled drop. Should this occur, the pipette must be washed and sterilized by boiling and the drops discarded; only after this can the pipette again be put into the eye-drop bottle. During drop instillation care should be taken not to turn the pipette upside down so that the solution runs into the rubber tube.

The frequency of drop instillation into the conjunctival sac depends on the type of drug, and on the character and course of the disease.

Ointments produce a more lasting effect than eye drops and are therefore preferred for certain diseases. Certain preparations insoluble or hardly soluble in water (yellow mercury, xeroform, synthomy-

cin) or emulsions (synthomycin) are also used in ointment form. Ointments are often used for burns of the eye to prevent the formation of adhesions between the dead conjunctiva of the eyelids and the eyeball conjunctiva. A good base for an eye ointment is a mixture of lanolin and vaseline in equal proportions. An ointment should be spread carefully to avoid irritation. A pea-sized portion of ointment is applied on a glass stick behind the lower eyelid (Fig. 28), the patient looking upwards. The patient is told to close his eye while the stick is moved towards the temple and withdrawn from the palpebral fissure; the ointment remains behind the eyelid. This is followed by light massage of the eyeball to spread the ointment uniformly over its surface.

Powders in the form of finely ground dust are applied to the conjunctiva of everted eyelids or upon the cornea with a glass stick (Fig. 29).

Massage of the eyelids. The margins of the lids are drawn together with the thumbs of both hands until they touch. They are compressed slightly by moving the fingers in the horizontal direction, or by inserting a flat glass stick into the conjunctival arch and pressing the margins of the lids against it (Fig. 30). Both procedures are effective for removing the secretion of the meibomian glands.

Painting the margins of the lids. A piece of sterile cotton is wound on the end of a glass stick, soaked in medicine and applied to the ciliary margin of the eyelid.

Subconjunctival injections—the introduction of a medicinal solution under the conjunctiva of the eye after anaesthetizing it by three instillations of a dicaine solution. This method is used to inject novocain, penicillin, streptomycin, dionine and other solutions, as well as oxygen.

The advantage of administering drugs by *electrophoresis* instead of subconjunctival injection is that it helps bring the ions closer

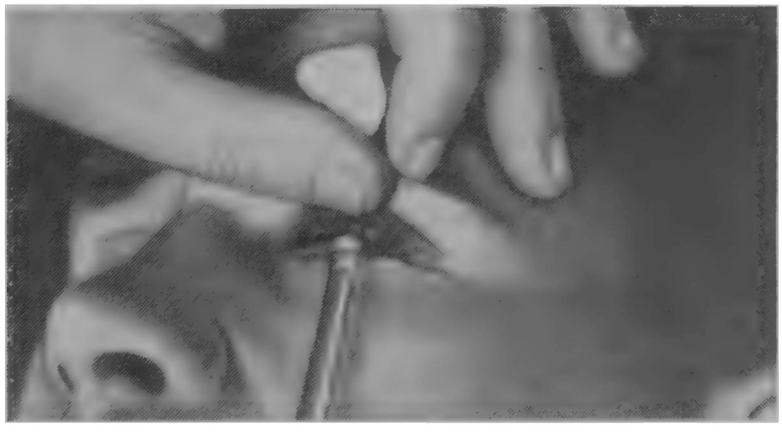


Fig. 30. Massage of the lids with a glass stick

to the focus of lesion; direct current also produces a favourable effect on the eye tissues.

Local drugs are subdivided according to their effect into astringents, caustics, disinfectants, resolvents, irritants, anaesthetics, analgetics, mydriotics and miotics. Many drugs can produce a combined or variable effect depending on their concentration. For example, weak solutions of zinc sulphate, blue vitriol (copper sulphate), and alum possess astringent properties while their strong solutions are caustic; yellow mercury ointment produce a disinfecting and resorbing effect, etc.

Astringent and Caustic Drugs

Astringent drugs constrict blood vessels, reduce hyperaemia of tissues, and relieve painful sensations. In strong concentration they produce a caustic and bactericidal effect.

Silver nitrate (Argentum nitricum) in a 1-2 per cent solution has more pronounced caustic than astringent properties. Formerly this substance was very widely used for acute conjunctivitis, trachoma, and in the prevention and therapy of gonoblennorrhea. Today it is being superceded by more effective drugs, such as sulphonamides and antibiotics which, in addition, produce none of the side-effects characteristic of silver nitrate: acute irritation causing pain and black discolouration of the conjunctiva (argyrosis).

Protargol and *collargol* are derivatives of silver and may therefore also cause argyrosis if used for a long time. Collargol is also employed for diagnosis of the condition of tear drainage.

Zinc sulphate (Zincum sulfuricum) is prescribed for conjunctivitis, corneal ulcers, and burns. A prescription of a strong solution (1-2 per cent) should be supplied with an exclamation mark to emphasize that there is no mistake. When zinc solutions are administered for some time their concentration should be increased because the patient becomes accustomed to this drug.

Antiseptic Drugs

Antiseptic drugs are prescribed when there are obvious signs of infection or for prevention of infection in acute conjunctivitis, ulcers of the cornea, keratitis, blepharitis, and trauma in the pre- and postoperative periods. These drugs are administered from 2 to 8-10 times daily and even more often if the patient's condition requires.

Quinine (chininum hydrochloricum) in a 1 per cent solution produces not only a disinfecting effect but also favours regeneration of the corneal epithelium and is, therefore, effective in healing lesions (erosions, corneal ulcers, burns and wounds). The solution

is prepared without addition of acid (sine acido paratum) and is instilled warm.

Mercury oxycyanide (hydrargyrum oxycyanatum), *boric acid* (acidum boricum), and *potassium permanganate* (kalium hypermanganicum) are used for eye irrigation.

Albucid (albucid natrium) is prescribed in solution or ointment form. Its efficacy is less than that of antibiotics.

Furacilin (furacilinum) is employed in solution and ointment form. It retains its properties upon prolonged storage and sterilization, and produces a disinfecting and tissue-regenerating effect.

Brilliant green (Brilliantgrün) is used for injuries to the skin of the eyelid, for painting the margins of the lid in blepharitis, and for antiseptic treatment of the operative field and the surgeon's hands for an operation.

Antibiotics: synthomycin or levomycetin ointments; solutions of these antibiotics; terramycin, tetracycline, biomycin ointments.

Penicillin and *streptomycin* are used in solution or ointment form. These solutions can also be introduced by injections under the conjunctiva or by means of electrophoresis in doses of 25,000 U once daily. It should be remembered that a penicillin solution retains its potency for 1-2 days, and a streptomycin solution for as long as 15 days.

Resolvents and Irritants

These drugs intensify blood and lymph circulation, favouring resolution of opacity of the cornea, haemorrhages, exudates and crystalline masses.

Yellow mercury ointment (hydrargyrum oxydatum flavum), in addition to its capacity to cause resolution produces a distinct antiseptic effect, and is used for opacity of the cornea, acute conjunctivitis, scrofulous conjunctivitis, keratitis and blepharitis.

Dionine (dioninum) is prescribed for prolonged treatment in the form of eye drops and ointments in increasing concentrations (from 1 to 10 per cent) so as to favour resolution of opacity of the cornea and vitreous body, remnants of crystalline masses and exudative foci in the choroid and retina. Dionine solutions are also injected subconjunctivally. The patient should be warned that dionine may cause a transient oedema of the eyelids and the mucous membranes, and also pain.

Potassium iodide (kalium jodatum) is used in the form of eye drops or injected by means of electrophoresis (3-5-10 per cent solution) if the above symptoms are present.

Glucose (glucosa, glucosum) in a 40 per cent solution is applied as eye drops or injected subconjunctivally (in doses of 0.5-1 ml), and in eye-baths for burns, keratitis, defects of the corneal tissue,

fresh opacities and as a healing and resolving agent. The application to the conjunctival sac of liquid honey is even more effective.

Vitamins in solution form are instilled into the conjunctival sac for initial cataract and persistent blepharoconjunctivitis. Burns, keratitis and ulcers of the cornea are healed by riboflavin, citral, ascorbic acid, and vitamin B_6. These drops are prepared on a 2 per cent glucose solution.

Anaesthetics

Cocaine (cocainum hydrochloricum) is an efficient anaesthetic but it may cause desquamation of the corneal epithelium (which, in turn, may produce ulceration of the cornea), dilation of the pupil and an increased intra-ocular tension. Therefore, anaesthesia is usually induced with dicaine which is free from certain unwanted properties of cocaine. Owing to its capacity to dilate the pupil, cocaine is often prescribed for iritis in combination with atropine.

Dicaine (dicainum) is used for anaesthetizing the conjunctiva and cornea during the extraction of foreign bodies from the cornea, in an examination of the intra-ocular tension and in surgery. Complete anaesthesia is induced by three drop instillations at intervals of 2-3 minutes.

Novocain (procaine) is injected subconjunctivally, subcutaneously, and retrobulbarly to induce anaesthesia in surgery and to block the sensory nerve branches in various diseases of the eye.

Mydriatics and Miotics

Mydriatics which dilate the pupil, are used in cases of iritis and iridocyclitis to lessen inflammatory symptoms, prevent the formation of adhesions between the iris and the crystalline lens or the cornea and to dilate the pupil during an examination of the ocular fundus and refractive power.

Atropine (atropinum sulfuricum) in solution or ointment form dilates the pupil and paralyzes accommodation. The pupil of the normal eye dilates 15 minutes after the application of atropine and remains dilated for a week or more. In certain cases of inflammation a grain of atropine has to be introduced into the conjunctival sac to dilate the pupil and keep it dilated for a certain time. The drug increases the intra-ocular tension and should therefore not be used for glaucoma or when an increased intra-ocular tension is likely. This should be kept in mind especially when atropine is given to patients older than 40 who are more frequently affected with glaucoma.

Atropine often causes dryness of the throat which is the initial symptom of poisoning. In certain patients who are hypersensitive

to this drug it causes eczema of the skin of the eyelids and the face, and conjunctivitis (atropine catarrh). Atropine drops are prescribed for children in a low concentration varying with their age (from 0.2 to 0.75 per cent). Their reaction to atropine is sometimes manifested by flushing of the face, a rise in the body temperature, and hallucinations. In such cases the drug should be withdrawn and replaced with another mydriatic. Usually these symptoms pass off without complications. In therapeutic practice a 0.1 per cent atropine solution is frequently used. Special precautions should be taken so that the patient does not accidentally drink a 1 per cent solution used in ophthalmological practice.

Scopolamine (scopolaminum hydrobromicum) produces an analogous though weaker effect than atropine, and is used in the same cases as atropine.

Homatropine (homatropinum hydrobromicum), *platyphylline* (platyphyllinum bitartaricum), *phenamine* (phenaminum or amphetamine sulphate) cause upon a single drop instillation a brief dilation of the pupil sufficient for examination of the ocular fundus and paresis of accommodation; in patients with a deranged regulation of the intra-ocular tension it is increased (phenamine is the safest drug in this respect).

Adrenalin (adrenalinum hydrochloricum) is used as eye-drops or for subconjunctival injections. It dilates the pupil, constricts the vessels and has a desensitizing effect. It is used in combination with atropine for diseases of the uveal tract, tuberculous and scrofulous affections, and various allergic diseases (spring catarrh, drug-induced blepharoconjunctivitis, etc.).

Miotic drugs cause spasm of accommodation, and lower the intra-ocular tension. They are prescribed mainly for glaucoma. Among these drugs are *pilocarpine* (pilocarpinum hydrochloricum) in solution or ointment form, *eserine* (eserinum salicylicum), *furamon* (furamonum), and *phosphacol* (phosphacolum) in solution. The effect of pilocarpine is different from that of the other miotics; therefore, when it fails to reduce the intra-ocular tension it is administered in combination with any of the above-mentioned drugs.

Corticosteroids

Cortisone—a hormone from the adrenal cortex extract—produces an antiallergic and antiphlogistic effect. It is used in the form of a suspension which is injected into the conjunctival sac 3-4 times a day, or subconjunctivally as a 2.5 per cent solution in a dose of 0.2 ml once in 3-4 days. It is prescribed for allergic diseases of the eye (spring catarrh, scrofulosis), keratitis (except herpetic and ulcerous forms), and for iridocyclitis, sympathetic inflammation, etc. Cortisone should be administered in combination with other anti-

Fig. 31. Monocular bandage

Fig. 32. Binocular bandage

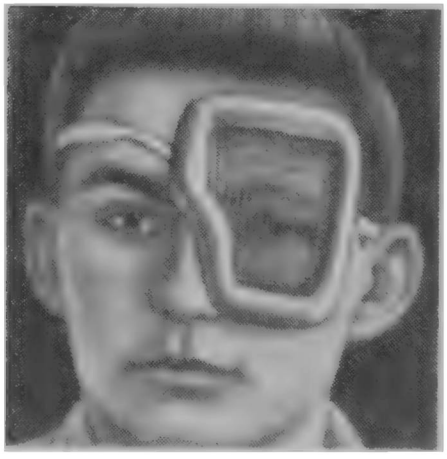


Fig. 33. Sieve-like patch on the eye

microbial drugs as a supplement to pathogenetic therapy. Other corticosteroids (prednisone, prednisolone) have greater potency than cortisone. Hydrocortisone ointment has recently been used on a growing scale. The effect of a 2 per cent pyramidon solution is analogous to that of cortisone.

Dressings

Dressings are applied to the eye after surgical operations, wounds and whenever the eye requires comfort and warmth. A dressing is contraindicated when there is a purulent or copious mucous discharge. Before a bandage is applied, the eye is patched with gauze and several layers of cotton-wool. It is sometimes sufficient to use a light dressing of one strip of bandage applied obliquely across the diseased eye with a turn about the forehead to fix the bandage in place. A better means of protecting the eye is a monocular dressing with several turns of bandage (Fig. 31). The bandage is applied obliquely from the side of the affected eye to the opposite side of the head, then across the occiput to the ear lobe, round the forehead and again across the eye, covering the inner side of the orbital region. The bandaging is completed with one or two turns round the head, with the knot on the forehead or on the temple on the side of the diseased eye.

A binocular dressing (Fig. 32) is applied as described above, by oblique turns of bandage covering alternately one eye and then the other. It is used to provide comfort for both eyes (after surgical extraction of cataract, transplantation of the cornea, operations for strabismus). In some cases, after the removal of a dressing or when

there is exudation, a metal net trimmed with gauze padding or a sieved metal plate is applied to the orbital region and secured in place with a tape tied round the head (Fig. 33). When the dressing is no longer necessary, a square gauze "curtain" is hung in front of the eye to shut out light. It is secured in place with a turn of bandage applied across the forehead or with a strip of adhesive plaster.

Dressings without bandage are simple and convenient; a gauze patch with cotton-wool is secured in place with adhesive plaster. A gauze patch with cotton-wool applied to the eye may be covered with a larger patch of gauze and glued with cleol (mastisol) to the cheek, temple and forehead skin. This type of dressing is widely used to cut off vision during the treatment of amblyopia in strabismus.

7

DISEASES OF THE EYELIDS AND LACRIMAL APPARATUS

DISEASES OF THE EYELIDS

Diseases of the Skin of the Eyelids

Because there is no fat in the subcutaneous tissue of the eyelid, haemorrhages, oedematous fluid, and air spread easily within it. Haemorrhages occur after injury to the eyelid, eyeball, orbit and skull. Fractures of the base of the skull and compression of the thorax are likely to cause haemorrhage in the depth of the palpebral tissue of both eyes, giving the appearance of a spectacles frame. Haemorrhages also occur in systemic diseases (scurvy, whooping-cough, diseases of the blood including haemophilia, chronic constipation, vascular hypertension). In view of the possibility of a simultaneous intra-ocular haemorrhage the visual acuity should be examined and, if it is impaired, the patient should be sent to an ophthalmologist. Haemorrhages quickly resolve. Treatment should be applied for the principal disease.

Subcutaneous emphysema usually occurs as a result of injury to and discontinuity of tissue in the inner wall of the orbit; air penetrates from the nasal cavity into the depth of the eyelid tissue, into the orbit or behind the eyeball. A characteristic symptom is a thickening and drooping of the eyelid simulating oedema. If pressure is applied there is a feeling of crepitation resembling the crackling of rumpled parchment-paper. If air penetrates into the orbit, the eyeball will protrude (exophthalmos). To identify the cause of emphysema and prescribe appropriate treatment, the patient should be seen by a rhinologist. A tight dressing on the eye is recommended, to facilitate quick expulsion of the air.

Oedema of the eyelids may be inflammatory, stagnant and non-inflammatory.

Inflammatory oedema is encountered with acute inflammatory conditions in the eyeball and its appendages, the orbit and the periorbital parts (stye, acute conjunctivitis, phlegmon of the lacrimal

sac or the orbit, inflammation of the lacrimal gland, ulcer of the cornea). It is accompanied by hyperaemia and oedema of the skin of the eyelid which is hot to the touch, and constriction of the palpebral fissure.

Oedema associated with congestive phenomena in the venous network of the lids and the orbit develops in a sharp spasm of the eyelids, thrombosis of the orbital veins and the cavernous sinus, and in orbital tumours. As a result of disturbance of the venous drainage, the skin of the eyelid becomes cyanotic, and the veins in the eyelid show clearly as thick tortuous vessels.

Noninflammatory oedema is found in diseases of the kidneys and the heart, with myxoedema, trichinosis, angioneurosis, etc. In view of the multiple causes producing such oedema, a thorough general examination of the patient and his organ of vision is required in order to identify the principal disease correctly and select the most effective method of therapy.

Inflammatory diseases of the skin of the eyelid are furuncles, eczema, erysipelas, abscess, phlegmon, anthrax, etc. Their clinical picture is identical to that produced by these diseases in other parts of the body.

Confined acute inflammatory conditions—furuncle of the eyebrow, abscess of the eyelids—are purulent boils attended by inflammatory oedema of the skin.

Erysipelas of the eyelids often spreads from the surrounding parts of the facial skin. It is characterized by a markedly circumscribed intense hyperaemia of the skin and by inflammatory oedema, fever, and swelling of the parotid lymphatic glands. Complication with orbital phlegmon is also possible.

Treatment. Sulpha drugs per os once every four hours or intramuscular penicillin injections. Local application of dry heat (hot-water bottles, sollux lamp); in erysipelas—irradiation of the eyelid and facial skin regions with ultraviolet light. If the eyelid abscess fails to open by pointing, it is incised if fluctuation is present. This operation is followed by the application of dressings with hypertonic salt solution or with synthomycin emulsion. It should be kept in mind that pus must not be expressed from the suppurative foci on the eyelids in view of the risk of spreading the infection through the venous network and causing thrombosis of the orbital veins and the cavernous sinus.

Diseases of the Margins of the Eyelids

Diseases of the margins of the eyelids run a chronic course and are prone to relapse. They occur with anaemia, malnutrition, hypovitaminosis, metabolic disorders, tuberculosis, scrofulosis, diabetes, chronic intoxication (helminthic invasions, constipation) as

Fig. 34. Ulcerative blepharitis

Fig. 35. Stye on the upper lid

Fig. 36. Chalazion of the upper and lower eyelids. View from the skin side

well as with diseases of the paranasal sinuses. Other important factors are uncorrected refraction anomalies, especially hypermetropia and astigmatism, unhygienic working and living conditions, and combinations of the above. The adverse effect of these factors may be aggravated by a local infection with the staphylococcus albus or staphylococcus aureus and streptococci.

Inflammation of the margins of the eyelids occurs in simple or squamous and ulcerative blepharitis (blepharitis simplex, squamosa and ulcerosa respectively).

Simple, or *squamous, blepharitis* (blepharitis simplex) is characterized by hyperaemia of the margins of the eyelids, deposition of delicate grey scales and crusts at the ciliary margin, falling out of the cilia, and itching. There is hypersensitivity to light, dust, and smoke; the eyelids are prone to turn red and swell.

Ulcerative blepharitis (blepharitis ulcerosa) causes moderate oedema of the palpebral margins and the formation of purulent scabs covering small ulcers (Fig. 34). The cilia are glued into bundles and fall out; some of them, as a result of cicatrization of ulcers assume a wrong position and rub against the eyeball. At the end of the disease, the palpebral margin without cilia cicatrizes and thickens, and the eyelid sometimes becomes everted.

Treatment. The primary measure is to eliminate the cause of the disease. Correction of refraction anomalies is also necessary. Polyvitamins are very helpful, especially cod-liver oil and riboflavin; tissue therapy is most beneficial.

Local applications involve painting the margins of the eyelid with antiseptic ointments or brilliant green solution twice daily. At the same time zinc phosphate or antibiotic solutions are instilled into the conjunctival sac for concurrent conjunctivitis. In cases of ulcerative blepharitis, the crusts are softened with ointment and removed, and then the ulcers are seared with brilliant green solution, iodine tincture or silver nitrate.

Meibomian blepharitis (blepharitis, blepharoconjunctivitis meibomiana) is a chronic or persistent relapsing inflammation of the margins of the eyelids and the mucous membrane. The disease produces itching, hyperaemia of the margins of the eyelids, a foamy discharge (changed secretion of the meibomian glands) accumulating at the angles of the palpebral fissure, and hyperaemia of the palpebral mucosa. A frequent complication is superficial infiltration of the cornea.

Treatment. Prolonged instillation of a zinc phosphate solution or a 3 per cent soda solution, massage of the margins of the eyelids with a glass stick to expel the contents of the glands and wiping of the margins with a piece of cotton-wool soaked in alcohol or ether. This is followed by painting the ciliary margin with an alcohol solution of brilliant green.

Stye (hordeolum) is a purulent inflammation of the hair-follicle or sebaceous gland at the root of the cilia (Fig. 35). The disease involves confined hyperaemia and tender swelling of the skin at the lid margin. A boil forms shortly, which after pointing discharges pus and particles of dead tissue. Styes are liable to relapse and often occur with blepharitis. Relapsing styes are due to the same causes as blepharitis; the patient requires a general examination and treatment.

Treatment. Very beneficial are autohaemotherapy and repeated blood transfusions, brewer's yeast, vitamins A,B_2, C, and in the acute period sulpha drugs per os. Local therapy involves the use of dry heat or UHF currents. As there is a risk of the condition spreading through the venous channels, ointments should not be rubbed into the margins of the eyelids and the stye should not be pressed. In the intervals between relapses of a stye the same local therapy as that used for blepharitis is helpful.

Chalazion is a chronic inflammation of a meibomian gland. It is shaped like a pea and felt with the fingers under the unchanged skin, and is visible under the conjunctiva (Fig. 36).

Treatment is surgical.

Changes in the Position and Movements of the Eyelids

Inversion of the eyelids develops as a result of cicatrization of the conjunctiva (*cicatricial entropion*) in trachoma, after diphtheria, burns, and pemphigus. Spastic entropion is due to a convulsive con-

traction of the orbicular muscle of the eyelid or its palpebral portion lying at the margin of the eyelid. Such an inversion is found in senile people with a flabby skin and chronic conjunctivitis, or in young people with a prolonged spasm of the eyelid; for example, in scrofulosis and inflammatory diseases of the anterior portion of the eye. In an inverted eyelid the cilia are turned towards the eyeball and rub against the cornea, causing irritation which may be the cause of its ulceration.

Treatment. Cicatricial entropion of the eyelids is treated by surgery, while spastic entropion requires treatment of the principal disease. Good results are produced by injections of a 1 per cent novocain (procaine) solution into the depth of the orbicular muscle of the eyelids or by attaching a drawn-off eyelid to the skin of the cheek with adhesive plaster. Where therapy fails, an operation to correct the position of the eyelid is indicated.

Eversion of the eyelids (ectropion) may be *cicatricial* as a result of burns of the lid and facial skin, severe disfiguring injuries, or anthrax; *spastic* in cases of spasm of the orbital portion of the orbicular muscle of the lid; and *paralytic* in cases of paralysis of the facial nerve supplying the orbicular muscle of the lid, or when its tone decreases due to senile flabbiness of the skin of the lids, in chronic conjunctivitis attended by hypertrophy of the mucous membrane, prolonged lacrimation, and maceration of the skin of the eyelid. In cases of ectropion, the margins of the eyelids are detached from the eyeball and the lacrimal punctum is not submerged in the lacrimal lake. as a result of which tear drainage is disturbed, tears flow down the cheeks and cause maceration of the skin, while the exposed conjunctiva dries up and becomes hypertrophied. The cornea may also become dry and subsequently ulcerous.

Treatment. In cases of cicatricial and paralytic ectropion surgery is used; in cases of spastic ectropion treatment is given for the principal disease, while individual cases require surgery.

Diseases of the muscles of the eyelids. The eyelids are moved by the orbicular muscle which closes the palpebral fissure, and by the levator palpebrae muscle which raises the upper lid. When these muscles are affected various disorders of movement of the eyelids result.

Blepharospasm—convulsive compression of the lids—is caused by reflex stimulation of the trigeminal nerve; it is usually encountered in inflammatory diseases of the anterior portion of the eyeball and is attended by excessive lacrimation and photophobia. These symptoms usually pass off when the patient recovers from the principal disease; sometimes blepharospasm persists for a longer time.

Treatment. In persistent spasm of the eyelids, it is helpful to supplement treatment of the principal disease with injection of a 5 ml dose of a 1 per cent novocain solution into the depth of the eyelid tissue, around the external palpebral commissure to the lower eyelid.

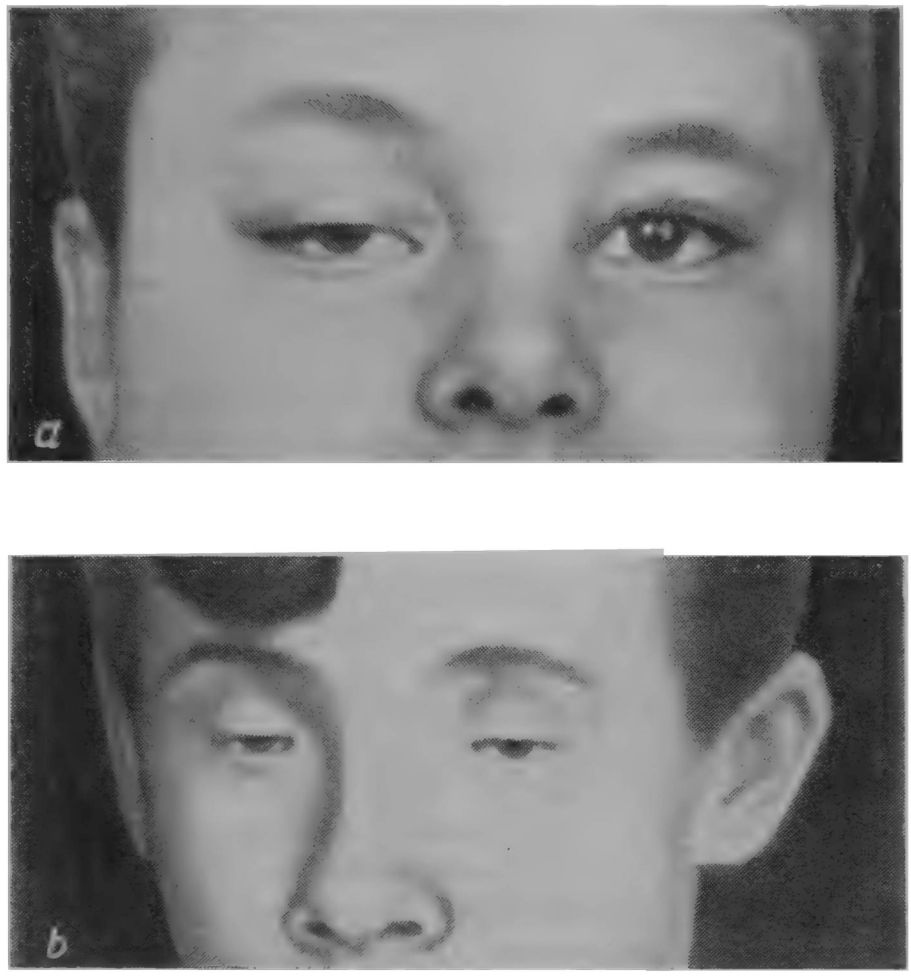


Fig. 37. Ptosis
a—unilateral; *b*—bilateral

Paralysis of the orbicular muscle due to paralysis of the facial nerve causes *lagophthalmos*, a condition in which the eyes cannot be closed and the palpebral fissure gapes (hare's eye). This results in disturbance of tear drainage and dryness of the cornea. Paralysis or paresis may develop after influenza, and in lesions of the nervous system and diseases of the middle ear. Lagophthalmos may also be associated with a considerable protrusion of the eyeball due to a retrobulbar tumour, as well as with thyrotoxicosis (Basedow's disease) and cicatricial changes of the eyelids.

Treatment. Elimination of the principal cause and prevention of the eyeball from drying by injecting into the conjunctival sac of fat (vaseline, cod-liver oil) or applying disinfectant ointments (penicillin, synthomycin). A persistent, irreversible condition requires an operation to suture the margins of the eyelids at the external and internal angles.

Drooping of the upper eyelid (ptosis) may be congenital or acquired (Fig. 37). The latter results from injury to the eyelid or pa-

ralysis of the oculomotor nerve, and diseases of the central nervous system. This is attended by other signs of affection of the oculomotor nerve (dilation of the pupil, disturbance of accommodation, paralysis of the muscles supplied by this nerve which rotate the eyeball). *Ptosis may be partial or complete.* In congenital ptosis, usually bilateral, a patient raises his eyelids by straining the occipitofrontal muscle, the forehead skin becoming wrinkled and the head tilted slightly backward. Sometimes a patient raises his eyelids with his fingers.

Treatment. In cases of acquired ptosis treatment should be aimed at eliminating the cause of the principal disease which is identified by a neuropathologist. In irreversible conditions and in congenital ptosis, surgery is indicated to increase the functioning of the muscle lifting the upper lid.

Moderate *ptosis* with recession of the eyeball into the orbit (*enophthalmos*) and contraction of the pupil (*miosis*) is noted when there is paralysis of the sympathetic nerve branch supplying part of the superior palpebral muscle (Mueller's muscle). This symptom is often a manifestation of disease of the nervous system.

Newgrowths (*tumours*) *of the eyelids* may be benign or malignant.

Atheroma is a benign newgrowth, a soft round formation which can be felt with the fingers under the skin of the eyelid at the external or internal angle of the eye.

Treatment. Extirpation of the tumour together with its capsule.

Angioma—a varicose dilation of vessels of the eyelid skin.

Treatment depends on the size and location of the tumour, and may include the following: neat-focus x-ray therapy, electrolysis, cauterization with carbonic acid flakes, surgery.

Malignant newgrowths—carcinoma, whose favourite site is the internal angle of the lower and, less frequently, of the upper eyelid. The disease is manifested by induration of the skin or a crater-shaped ulcer with dense margins, a knobbly bed with disintegrating tissue, bleeding surface and cicatricial portions. The tumour may extend to the paranasal sinuses, the orbit, and the eyeball. Metastases into the lymphatic glands and distal organs are observed rarely and at advanced stages of the disease.

Treatment. Radiation therapy, or surgery (excision of the tumour, and in far-advanced cases, of the eyeball and orbital contents followed by radiation therapy) should be undertaken as early as possible.

DISEASES OF THE LACRIMAL APPARATUS

Dacryoadenitis, inflammation of the lacrimal gland, is encountered rarely. In its acute form the disease is usually manifested by a bilateral redness and tenderness of the skin of the eyelid at the

external angle, and swelling and drooping of the eyelid, mainly externally. When the eyelid is drawn off and the patient looks inwards, a swollen and inflamed gland protrudes from under the external orbital margin. Acute dacryoadenitis develops as a complication of infectious diseases, such as influenza, enteric fever, pneumonia, tonsillitis, and paratyphoid.

Treatment. Transthermia with UHF currents, dry heat or warm dressing; sulpha drugs per os, penicillin by intramuscular injection. When there is suppuration of the gland, an additional incision is sometimes made on the side of the skin where the fluctuation is most marked, and suction dressing is applied.

The Lacrimal Passages and Their Diseases

The principal sign of disturbed functioning of the lacrimal apparatus is *lacrimation*, tears overflowing the lower margin of the eyelid and running down the cheek. Lacrimation is especially severe in cold and windy weather.

The causes of lacrimation vary. Most frequently lacrimation is caused by obstruction of the lacrimal passages due to a change in position, atresia or contraction of the lacrimal punctum, the canaliculus, and the lacrimal sac proper at the site of its communication with the nasolacrimal duct or at the duct outlet.

Dislodgement of the lacrimal punctum as a result of its eversion is encountered in cases of scars of the skin of the eyelid after burns, at the outcome of ulcerative blepharitis, in cases of senile flaccidness of the skin of the eyelid, and chronic conjunctivitis with hypertrophy of the mucous membrane.

Changes in the diameter and position of the lacrimal punctum are seen during examination by the conventional methods; obstructions of the other portions of the lacrimal passages are detectable by special procedures: the collargol test, irrigation of the lacrimal passages, probing, x-ray photography.

The collargol test is performed as follows: a 2 per cent collargol solution is instilled by drops into the conjunctival sac; in two minutes it becomes absorbed into the lacrimal canaliculi and disappears from the conjunctival sac. When pressure is applied to the region of the lacrimal sac, a drop of collargol is expressed from the lacrimal punctum, which indicates normal functioning of the lacrimal punctum and the canaliculus. This is known as *the positive canaliculus test.* If collargol remains in the conjunctival sac longer than 2-5 minutes or overflows the eyelid margin with tears and fails to be discharged from the lacrimal punctum when pressure is applied to the lacrimal sac region, this indicates that the lacrimal punctum and the canaliculus are not functioning correctly, i.e., *the canaliculus test is negative.*

Fig. 38. Irrigation of the lacrimal passages

To determine the permeability of the sac and nasolacrimal duct, *a nasal collargol test* is made. For this purpose, a cotton-wool tampon is inserted into the inferior nasal meatus and collargol is instilled into the conjunctival sac. If the lacrimal passages are clear, the cotton-wool will become stained with collargol in five minutes' time (*positive test*). If the lacrimal passages are obstructed, the tampon will not stain (*negative test*). The nasal collargol test may be carried out in a simpler way: the patient is told to blow his nose, each nostril separately, five minutes after collargol instillation. If the lacrimal passages are clear, the nasal discharge will take a stain.

The site of constriction or obstruction of the lacrimal passages is found by means of irrigation and probing. After preliminary anaesthesia of the conjunctiva by three instillations of dicaine solution the lower lacrimal punctum is dilated with a cone-shaped probe, and 5-10 ml of physiological saline solution are introduced into the lacrimal canaliculus with a syringe through a special cannula or a common needle with a blunt end (Fig. 38). With normal permeability of the lacrimal passages the liquid flows out of the nose in a stream, but when they are constricted it flows out in drops and when they are completely obstructed it flows back through the superior or inferior lacrimal punctum.

Probing (Fig. 39, *1*, *2*, *3*) is used both for diagnosis and, in individual cases, for therapy to dilate the lacrimal passages, e.g.,

in the newborn and infants with dacryocystitis and sometimes in adults.

The procedure is carried out with Bowman's probe of variable thickness. The probe is inserted through the inferior lacrimal punctum and passed first in the vertical and then in the horizontal direction as far as the nasal bone; after this, the free end of the probe is turned upwards and, keeping to the bone, the probe is passed through the lacrimal sac and the nasolacrimal duct into the nose. This manipulation should be performed by a specialist, because it requires great skill and involves the risk of forming a false passage and other complications.

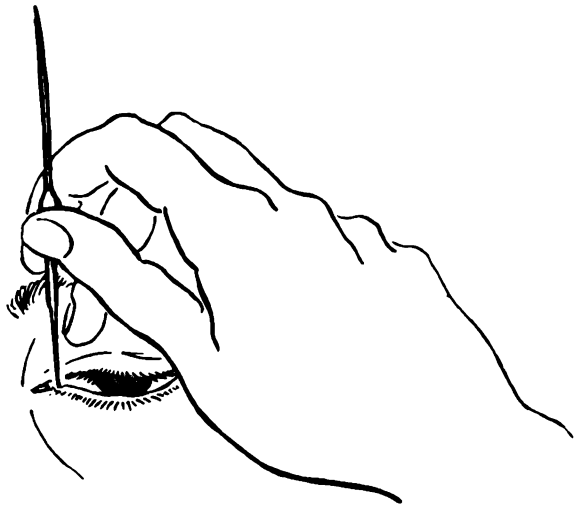
A more accurate and safe method for identifying the condition of the lacrimal passages is x-ray photography following the introduction of a contrast stain into the lacrimal sac.

Inflammation of the Lacrimal Sac

Dacryocystitis (dacryocystitis chronica) is a chronic inflammation of the lacrimal sac. This disease develops as a result of stricture or obstruction of the lacrimal passages due to a constant stasis of tear secretion in the sac causing infection with microbial flora. The major symptoms of this disease are lacrimation and pus discharge from the lacrimal sac, increasing when pressure is applied to its surrounding parts. Constant discharge of pus into the conjunctival sac leads to chronic conjunctivitis and involves the risk of a purulent ulcer of the cornea forming if it is injured, however slightly. Such ulcers are a serious danger to vision and often cause the wall-eye, i.e., leukoma of the cornea with partial or total loss of vision.

To prevent the risk of chronic dacryocystitis developing in patients with lacrimation (particularly, when the collargol test is negative), the medical assistant or nurse is obliged to send such patients to an ophthalmologist for examination. This is especially urgent in cases of manifest purulent dacryocystitis which can easily be identified by pus discharge from the lacrimal puncta when pressure is applied to the peripheral tissues of the lacrimal sac. Bearing in mind the significance of dacryocystitis in the formation of corneal ulcers, pressure should always be applied to the region of the lacrimal sac, even if the patient has only complained of a superficial injury to or a foreign body in the cornea. In cases of pus discharge or complaints of incessant lacrimation the patient should immediately be sent to an ophthalmologist.

Treatment. Dacryocystorhinostomy is an operation for forming a communication between the lacrimal sac and the nasal cavity. In elderly persons and in cases of rough changes of the sac, the latter is excised. The advantage of dacryocystorhinostomy over



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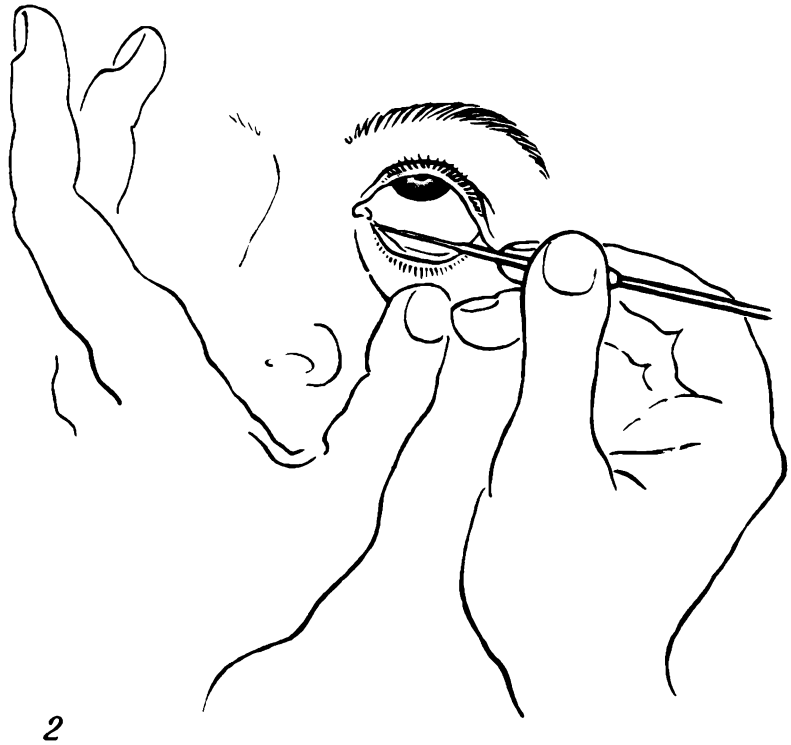

2

excision of the lacrimal sac is that it does not lead to disorders in tear drainage.

Acute inflammation (phlegmon) of the lacrimal sac (dacryocystitis acuta or phlegmona sacci lacrimalis) (Fig. 40) occurs in chronic dacryocystitis when the suppurative process extends to the cellular tissue surrounding the lacrimal sac. The disease manifests itself by marked redness, swelling and tenderness of the skin in the sac region, malaise, and fever. These are gradually followed by soften-

Fig. 39. Probing of the lacrimal passages

1—insertion of probe into the lacrimal punctum;

2—insertion of probe into the lacrimal canaliculus;

3—insertion of probe into the lacrimal sac

ing of tissues, fluctuation, and pointing of the phlegmon and pus discharge. A phlegmon may give rise to a fistula constantly discharging pus and to tear secretion. Sometimes the disease ends with gradual resorption of the infiltrate and pus discharge through the lacrimal puncta.

Treatment. Heat, transthermia with UHF currents, irradiation with a quartz lamp, sulpha drugs per os, or antibiotics intramuscularly. When there is fluctuation the boil is opened along the

Fig. 40. Phlegmon of the lacrimal sac

course of the lacrimal sac. Since relapses of this disease are inevitable, the patient should undergo one of the above operations, preferably when the acute symptoms abate. Such a patient should naturally be sent to an ophthalmologist for consultation and further treatment.

Dacryocystitis in the newborn is characterized by lacrimation and pus discharge from the lacrimal puncta, particularly when pressure is applied to the region of the lacrimal sac.

Treatment. This should be started immediately after the diagnosis has been made. First, massage of the region of the lacrimal sac is recommended. The fingers should be pressed downwards from above so as to express pus into the nasal cavity through the nasolacrimal duct rather than through the lacrimal puncta. Disinfecting eye drops should be applied at the same time (penicillin or collargol). If a massage given for several days fails to produce an improvement, the lacrimal passages should be probed so as to restore tear drainage. This manipulation should only be entrusted to an ophthalmologist; the medical assistant or nurse should therefore send the patient in good time to a specialist, bearing in mind that dacryocystitis of the newborn may become aggravated with phlegmon of the lacrimal sac and that constant pus discharge may bring about a number of severe complications.

8

DISEASES OF THE CONJUNCTIVA

Diseases of the conjunctiva are quite common. The most frequent of them is inflammation of the conjunctiva known as *conjunctivitis.* According to their causative factors, these diseases are subdivided into the following three basic groups:

1. *Conjunctivites of exogenous origin.* These comprise inflammations due to infectious diseases, and harmful physical and chemical agents, usually of occupational character.

2. *Conjunctivites of endogenous origin* associated with systemic diseases (metastatic, allergic).

3. *Degenerative changes of the conjunctiva.*

Depending on their course, conjunctivites may be *acute* or *chronic.*

Patients with acute conjunctivitis, which usually comes on suddenly, complain of an itching sensation in the eyes like that caused by a foreign body, and of lacrimation, photophobia, and exudation. Physical examination reveals hyperaemia, oedema of the conjunctiva of the eyelids and palpebral folds, a varying amount of exudation and sometimes false membranes on the tarsal mucosa. Oedema and spasm of the eyelids, lacrimation and photophobia are often present, depending on the intensity of the inflammatory process and its aetiology. Acute conjunctivites may become complicated with a corneal lesion.

When the conjunctiva is examined attention should be given to the character of redness—hyperaemia of the conjunctiva; differentiation should be made between *superficial* or *conjunctival* hyperaemia and *deep* or *pericorneal* hyperaemia. The former is characteristic of disease of the conjunctiva proper, while the latter is a sign of affection of the cornea, the iris and the ciliary body.

Conjunctival hyperaemia is due to excessive blood in the superficial vessels, and is manifested by redness of the palpebral conjunc-

tiva, the fold and the eyeball conjunctiva continuous with it. The superficial bright-red vessels, dilated and engorged with blood, are clearly visible. Displacement of the scleral conjunctiva by the lid causes displacement of the entire vascular network.

Pericorneal hyperaemia is due to the dilation of the deep-lying vessels (anterior ciliary). These vessels visible through the tissues lend a cyanotic hue to the conjunctiva around the limbus. Displacement of the conjunctiva does not cause displacement of the vessels. Both types of conjunctival injection may occur in combination. This is known as mixed hyperaemia and is found together with a disease of the palpebral and bulbar conjunctiva. The ability to identify the character of hyperaemia is very important for differential diagnosis between diseases of the conjunctiva and those of the eyeball.

CONJUNCTIVITES OF EXOGENOUS ORIGIN

A. Acute Conjunctivites

The onset of acute conjunctivitis is preceded by a brief incubation period. The disease is often bilateral, but may affect one eye first and then extend to the other, should treatment be delayed or neglected. Acute conjunctivitis is caused by various pathogenic microbes (Koch-Week's or diphtheria bacillus, pneumococcus, gonococcus, Morax-Axenfeld's diplobacillus, etc.). Conjunctivites of the above-mentioned aetiology have specific features depending on a particular causal agent.

Acute contagious (epidemic) conjunctivitis. This disease, commonly called pink-eye and known as Koch-Week's acute contagious conjunctivitis, is encountered chiefly in summer and autumn, and in hot climates it becomes epidemic. The disease is caused by Koch-Week's gram-negative bacillus which can be identified by bacterioscopic examination of the conjunctival discharge in sick persons. The most characteristic signs of the disease are oedema of the inferior palpebral fold (Fig. 41), hyperaemia of the conjunctiva and a predominantly purulent discharge. Other frequent symptoms are subconjunctival haemorrhages (Fig. 42), and the formation of false membranes, like those seen in diphtheria, on the eyelid conjunctiva.

In addition, there are mild forms of the disease with slight hyperaemia and a scanty discharge. Conjunctivitis, particularly in infants, often gives general symptoms resembling those of influenza (running nose, fever, insomnia). Koch-Week's conjunctivitis is more frequently found in children, chiefly those of a very young age, especially in debilitated children with scrofulosis, intestinal diseases, and vitamin deficiency. It often extends to the cornea

Fig. 41. Oedema of the inferior palpebral fold

Complications, however, may arise in adults also, in the form of infiltrations and corneal ulcers which are likely to produce a wall-eye with all attendant developments. The disease is highly contagious, and is conveyed by flies settling on objects in general use, on the eyelids of sick persons and then on those of healthy ones. It can also be spread by the hands touching a sick and then a healthy eye. The spread of the disease is favoured by poor sanitary and hygienic conditions.

Treatment. Penicillin, synthomycin, albucid (sulphanil acetamide), furacilin (5-nitro-2-furaldehyde semicarbazone) are applied locally in solution form. It is recommended that these drugs be instilled into the conjunctival sac from four to six times daily. Experience has proved that the best effect is produced by sulpha drugs taken per os round the clock once in every four hours over three to five days (until the cessation of discharge). This therapy aids recovery, and pathogenic organisms disappear, which lessens the danger of the infection spreading and obviates the need to put the patients in quarantine. Timely and rational therapy, personal hygiene and vigorous control of flies are exceedingly important for success in fighting epidemics of Koch-Week's conjunctivitis.

Fig. 42. Subconjunctival haemorrhage

Pneumococcic conjunctivitis is caused by a gram-positive diplococcus (pneumococcus). It is most commonly encountered in children with pneumonia. Acute initial

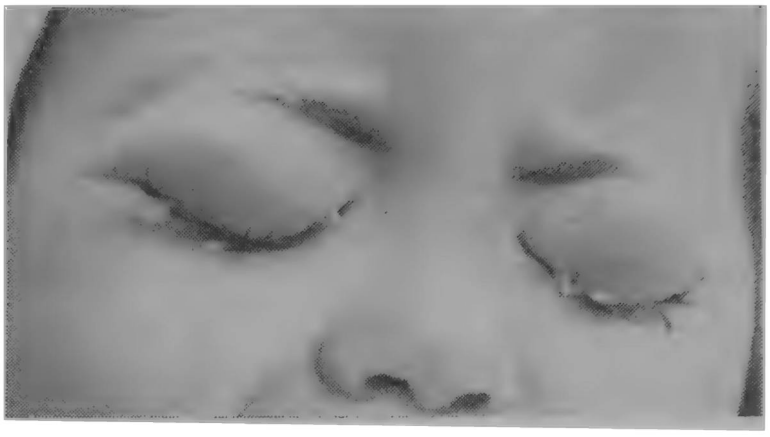

Fig. 43. Gonorrheal conjunctivitis

symptoms of the disease may appear in one eye and soon extend to the other. The patient has a severe palpebral oedema, a copious purulent discharge, and false membranes on the tarsal mucosa which are easily removable with a piece of cotton-wool. A marked improvement comes in a few days' time. In other cases the disease may take a longer course with different symptoms: redness of the margins of the eyelids, severe oedema of the eyeball conjunctiva, scanty discharge, and sometimes marginal infiltrations or ulcers of the cornea.

Diagnosis is based on the clinical picture of the local and systemic disease and verified, whenever possible, by laboratory examination of the conjunctival discharge.

Treatment. Frequent instillation of penicillin solution or retropalpebral application of synthomycin or levomycetin ointment in combination with intramuscular penicillin injections or biomycin per os.

Diplobacillary conjunctivitis (Morax-Axenfeld's disease) is the most frequent form of conjunctivitis and runs a subacute and chronic course. It is caused by Morax-Axenfeld's diplobacillus. The characteristic symptoms of this disease are redness and itching at the external angles of the palpebral fissure, where examination often reveals hyperaemia, maceration and fissures of the skin. The palpebral conjunctiva is moderately hyperaemic, and the discharge is scanty and mucopurulent. The disease may be accompanied by greyish infiltrations in the cornea on the border with the limbus.

Treatment. Specific treatment is with solutions of zinc sulphate which are instilled into the eye three to four times daily; the eyelid margins are painted at the angles with zinc oxide ointment. The treatment is highly beneficial and favours early recovery. As the disease is prone to relapse it is recommended that the zinc sulphate drops continue to be instilled for ten to fourteen days, even after obvious recovery.

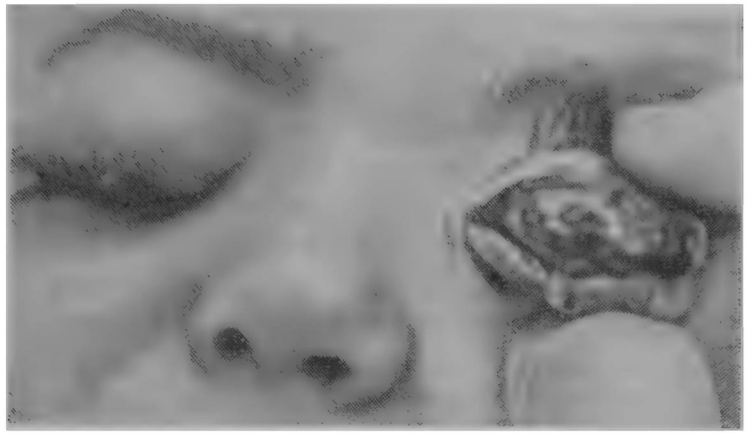

Fig. 44. Diphtheritic conjunctivitis

Gonoblennorrhea. This disease is due to the Neisser gonococcus, a gram-negative diplococcus present inside and outside leucocytes. Gonoblennorrhea occurs in the newborn, infected from their mothers at birth, in children acquiring infection from their mothers or nursing personnel, and in adults. In the latter case, it develops in patients with gonorrheal urethritis or when pus is conveyed from a sick person to a healthy eye, for example, in medical personnel. The disease gives characteristic symptoms irrespective of the sufferer's age.

One or two days after infection the eyelids become markedly oedematous and indurated, with hyperaemia of the skin and a scanty sero-sanguineous secretion. In two or three days' time, the tissues of the eyelids become softened and a copious purulent discharge appears (Fig. 43). The conjunctiva is hyperaemic, loosened in structure and markedly oedematous. The oedematous eyeball conjunctiva forms a circular swelling around the cornea. Ulcers are also likely to occur on the cornea and, spreading over its surface and into its depth, sometimes produce a severe leukoma of the cornea (wall-eye) which may result in total blindness or may even destroy the eyeball. In tzarist Russia, 4 per cent of the blind lost their vision owing to this disease. Gonoblennorrhea in adults presents an analogous clinical picture but runs a more severe course, and is often complicated with lesions of the cornea.

Diagnosis of gonoblennorrhea is verified by bacterioscopic examination of discharged matter in which gonococci are found. One should take into account the possibility of gonorrhea-like conjunctivites of non-gonorrheal aetiology (due to pneumococci, Koch-Week's bacilli, adenoviruses, or following cauterization of the mucosa with silver nitrate). Gonoblennorrhea in the newborn is prevented by means of silver nitrate (A. Matveyev, 1853; Krede, 1881). Immediately after birth the infant's eyelids are wiped with cotton-wool soaked in boric acid solution, which is followed by

instillation of one or two drops of 2 per cent silver nitrate solution into the conjunctival sac. This method has a number of disadvantages: failure to prevent the disease in isolated cases, the very irritating effect of the preparation on the eyeball conjunctiva and the risk of an eye burn (if a 10 per cent solution is instilled by mistake instead of a 2 per cent solution). Fresh silver nitrate solutions should always be at hand and kept in dark glass stored away in a dark place lest they should lose their medicinal properties.

Recently gonoblennorrhea has been effectively prevented using a fresh penicillin solution which is instilled into the infant's eyes twice—immediately after birth and two hours later.

Experience has shown that a 1 per cent synthomycin emulsion administered by the same methods is even more beneficial; its advantage is that it has a greater antiseptic effect and retains its properties for as long as one year.

Prophylaxis of gonoblennorrhea in adults requires early diagnosis and therapy of specific urethritis. Personal hygiene is necessary so that the patients do not infect the conjunctiva by contamination with the hands.

Mothers of children with gonoblennorrhea should be examined immediately and, should gonorrhea be found in them, subjected to appropriate therapy.

Treatment. The remedies used today are intramuscular injections of penicillin, streptomycin or administration of sulphonamides per os. This therapy can be combined with frequent instillations of antibiotic eye drops. Treatment results in early elimination of gonococci from the conjunctival discharge and recovery, and also prevents complications in the cornea.

Diphtheria of the conjunctiva. This disease is caused by the Loeffler bacillus. In typical cases the first signs are hyperaemia and severe oedema of the eyelids. The latter become so indurated that they cannot be everted for examination of the conjunctiva.

A scanty muco-purulent discharge appears at the same time. In two or three days' time, the eyelid tissues soften and the discharge becomes more profuse. Eversion of the eyelids reveals on the conjunctiva necrotic false membranes of the cartilage which are removed with difficulty leaving an ulcerated surface (Fig. 44). Other common signs are necrotized patches on the palpebral skin and oedema of the eyeball conjunctiva.

Within a week of the commencement of therapy, the discharge becomes more copious, the false membranes fall off, and the conjunctival lesions turn into a granulated surface which gradually heals to leave stellate scars. Sometimes adhesions form between the conjunctiva of the eyeball and that of the lids. Diphtheria of the conjunctiva may become complicated by a corneal ulcer producing leukoma (wall-eye) and in the worst cases may result in destruction of the eye.

The above form of the disease is called *diphtheritic* as distinct from the milder, *croupous* form characterized by moderate oedema of the eyelids and the eyeball conjunctiva, and a scanty discharge and deposition on the tarsal conjunctiva of delicate, superficial false membranes which are easily removed, exposing a bleeding mucosal surface. The cornea is usually unaffected, and the disease has a favourable outcome. Diphtheria of the conjunctiva is commonly encountered in children and belongs to the group of rare localizations; a lesion of the conjunctiva is usually attendant on diphtheria of the pharynx, nose and larynx. It may, however, develop as an independent disease accompanied by the same symptoms of intoxication and complications as are seen in diphtheria of the air passages.

It should be borne in mind that the above picture of diphtheria may be caused by other pathogenic organisms, such as Koch-Week's bacilli, pneumococci, streptococci, etc., particularly in debilitated children after infectious diseases. In addition, atypical manifestations of diphtheria are also likely to occur (above all, in vaccinated children) in the form of catarrhal conjunctivitis.

Diagnosis is based on the detection of diphtheria bacilli in a smear and in a culture of the conjunctival discharge. Note is taken of the patient's general condition and other symptoms of the disease, as well as the epidemiological situation, particularly in children's institutions.

Treatment. In view of the gravity of the disease, patients suspected of having diphtheria are placed in the isolation ward of an infectious disease hospital without waiting until the results of laboratory analysis are known. They are given an injection of antidiphtheritic serum in a dose of from 6,000 to 10,000 Units.

In grave cases it is recommended that serum injections are combined with general antibiotic therapy. Heat is applied locally for palpebral oedema, the eyes are irrigated with potassium permanganate solution and synthomycin ointment is applied later. If the cornea is involved, the same therapy is required as for corneal ulcers.

Acute conjunctivitis of adenovirus aetiology is concurrent with catarrh of the upper air passages, and may give various clinical signs. *Catarrhal* conjunctivitis is marked by mild hyperaemia of the palpebral conjunctiva and a scanty discharge, while *membranous* conjunctivitis resembles the specific or croupous forms of diphtheria. Its characteristic signs are catarrh of the upper air passages, high fever, seasonal incidence, chiefly among nurslings and small children, inefficacy of antidiphtheritic serum, absence of other symptoms of diphtheria, and negative laboratory tests for Loeffler's bacilli.

Conjunctivitis also occurs in a *follicular* form clinically similar to trachoma.

Keratites complicating the above-described diseases and keratitis therapy are discussed on p. 121.

B. Inflammation of the Conjunctiva Due to Mechanical, Chemical and Physical Factors

Because of the prolonged effect of irritating factors often associated with the patient's occupation (dust, intense light, chemical agents) these conjunctivites usually run a chronic course.

They are often encountered in conjunction with uncorrected refraction anomalies, blepharites, dacryocystitis, diseases of the paranasal sinuses, metabolic disorders, vitamin deficiency, helminthic invasions, chronic constipation, etc. The usual complaints are heaviness in the eyelids, burning pain and sensation of a foreign body in the eyes, lacrimation, photophobia, exudation (particularly in the morning), and inability to work long because of visual fatigue. Examination of the eyes reveals hyperaemia of the conjunctiva, sometimes looseness of its tissues, and a small amount of exudation.

Treatment of these conjunctivites is beneficial provided the cause of the disease has been identified. Refraction anomalies should be corrected, a surgical operation should be undertaken for dacryocystitis, and the appropriate therapy should be given for blepharitis. Solutions of zinc sulphate, penicillin, synthomycin, albucid, furacilin are applied locally, as well as synthomycin or levomycetin ointments. If the disease is associated with occupational hazards or poor sanitary and hygienic conditions, the necessary measures should be taken to improve them. In individual especially persistent cases of occupational conjunctivitis, it may be necessary for the patient to change his trade.

Another occupational disease of the conjunctiva is *electrophthalmia*. It occurs in electric welders if their eyes are inadequately protected against ultraviolet light and in film actors whose eyes are exposed to intense light during filming. The disease comes on within five to six hours of irradiation, and is manifested by intense photophobia, lacrimation, pain in the eyes and oedema of the eyelids.

Treatment. Eye lotions, instillation of dicaine and adrenalin, cortisone and pyramidon solutions. All symptoms usually disappear within a few hours.

Conjunctivites of Endogenous Origin

Measles conjunctivitis. Conjunctivitis is the initial symptom of measles. It becomes manifest several days before the eruption of a skin rash. Measles conjunctivitis, developing under the influ-

ence of a virus infection in a debilitated child's body, may become complicated with a corneal ulcer likely to produce leukoma (wall-eye).

Treatment. A child with measles should have its eyes regularly irrigated with boric acid or potassium permanganate solution. Disinfectant solutions of synthomycin or furacilin are administered by drop instillation three to five times daily; antibiotic ointments are also applied. It is very important to watch the condition of the cornea. In the presence of a purulent discharge or marked symptoms of conjunctivitis it is helpful to prescribe sulpha drugs per os.

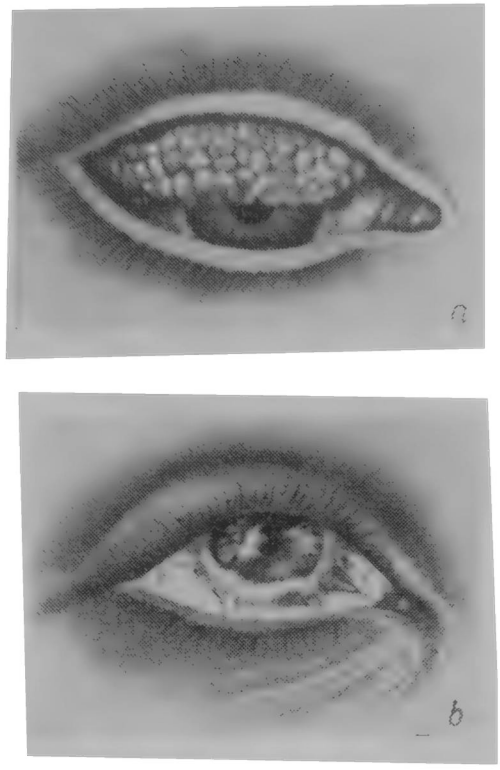

Fig. 45. Spring catarrh
a — conjunctival form; *b* — limbal form

Spring catarrh is a chronic disease of the palpebral conjunctiva usually encountered in boys. It has a seasonal incidence, recurring yearly with the coming of sunny days in spring. The disease chiefly begins at school age and ends at puberty. The characteristic complaints are itching, particularly at the angles of the palpebral fissure, and of photophobia. The superior tarsal and the unchanged palpebral conjunctiva are covered by large dense eminences or follicles the disposition of which resembles a "cobblestone pavement" (Fig. 45*a*). Sometimes, such proliferations are found at the limbus (limbal form) (Fig. 45*b*). The palpebral fold is unchanged. A characteristic sign of spring catarrh is the presence of eosinophils in a scraping from the conjunctiva. The aetiology of the disease is obscure; it is attributed to vitamin A deficiency, endocrinic insufficiency, hypersensitivity to ultraviolet light, and allergy.

Treatment. Peroral administration of cod-liver oil, riboflavin, calcium chloride, intramuscular injections of aloe extract. Zinc sulphate with adrenalin is applied locally. Drop instillations of cortisone and a 2 per cent pyramidon-adrenalin solution are especially beneficial. Protective dark glasses should be worn. In individual cases which do not respond to therapy, moving to another climate aids recovery.

Conjunctivites caused by drug allergies. These are due to prolonged administration of atropine, eserine, penicillin, streptomycin and other drugs. However, inasmuch as such conjunctivites are due to hypersensitivity to these drugs, they may develop even after a single drop instillation. Such conditions have been noted in med-

Fig. 46. Pterygium

ical personnel regularly handling antibiotics. The objective signs are oedema, hyperaemia, dryness or, on the contrary, maceration of the skin of the eyelid. The palpebral conjunctiva is markedly hyperaemic, and follicles are sometimes noted. If the drug responsible for the sickness is withdrawn the patient soon recovers.

Scrofulous conjunctivitis (see p. 120).

DEGENERATIVE CHANGES OF THE CONJUNCTIVA

Pingueculae. These are small triangular yellowish patches of degenerative connective tissue, which form upon the conjunctiva in the palpebral fissure between the limbus and the palpebral angle. Shaped like a small eminence of a yellowish colour, pinguecula does not grow in size, and is not dangerous to the eye and vision. This fact should be explained to patients who are often worried by the appearance of this small "tumour" in the eye.

Treatment is not required.

Pterygium (wing-shaped membrane) is a triangular patch of mucous membrane growing into the superficial layers of the cornea, more frequently from the inner side of the limbus (Fig. 46). The membrane tends to grow in size and may extend as far as the centre of the cornea, which may cause a considerable loss of vision. Pterygium is usually encountered in regions with a hot climate, plentiful sunshine and dust irritating the eyes. The aetiology of the disease is uncertain.

Treatment consists in surgical removal, which, however, may be followed by a relapse.

9

TRACHOMA

Trachoma is a chronic contagious inflammation of the eyelid conjunctiva with marked sub-epithelial infiltration, formation of follicles (granulations), disintegration and degeneration of the latter followed by cicatricial contraction. The clinical course of trachoma is characterized by a superficial diffuse inflammatory vascularization of the cornea known as *pannus*. In individual cases the disease may take an acute course from the outset.

THE CLINICAL PICTURE AND DIAGNOSIS OF TRACHOMA

The clinical picture of trachoma is variable, and depends on its extension, the intensity of granulations and infiltration, and the character of complications. The disease may run a mild, a moderately severe or a severe course. The course of the disease largely depends on the patient's general condition, and is more serious in scrofulosis, tuberculosis, malaria, helminthic invasions, metabolic disorders, and vitamin deficiency, as well as in intercurrent local infections. Another important factor is therapy, in particular its promptness, regularity, and individual selection of rational treatment and medical drugs.

Trachoma runs a characteristically protracted course, lasting for years as a result of exacerbations, relapses and complications. In addition, the disease may give vague, atypical manifestations which yield early to therapy, have a favourable outcome and sometimes pass off spontaneously (particularly in children).

The course of trachoma is known to have four stages. *The first stage*—trachoma I (Fig. 47*a*)—is progressive and usually begins inconspicuously. It is commonly encountered in children, and is

often identified during a prophylactic examination or when a patient is admitted complaining of exudation from and burning pain in the eyes. At the onset of the disease there is hyperaemia and infiltration (swelling) of the palpebral folds, especially the upper one. The follicles, shaped like dull-grey (sago-like) elevations, are scattered in disorder within the depth of the conjunctiva of both palpebral folds. Later, follicles erupt on the epicanthus, at the inner canthus, and on the tarsal conjunctiva (follicular form).

The growth of follicles on the tarsal conjunctiva may be accompanied by hypertrophy of the submucous tissue, the conjunctiva having a peculiarly corrugated and velvety surface (papillary form). Follicles may also appear on the eyeball conjunctiva by extension from the palpebral fold to the limbus as well as to the lacrimal caruncle. The presence of discharge is typical of cases with an intercurrent secondary infection and contributes to the spread of trachoma in the patient's surroundings.

Infiltration of the tarsal cartilage and its attached superior palpebral levator sometimes results in drooping of the upper eyelid. The first stage of trachoma may continue indefinitely.

The second stage—trachoma II (Fig. 47*b*)—is a regressive form. with degeneration and necrosis of certain follicles, degeneration of cells of the inflamed submucous tissue and formation of individual scars, along with the eruption of new follicles. If there is a very extensive growth of granulations in the palpebral fold and on the tarsal conjunctiva, they degenerate and coalesce into a jelly-like mass (gelatiniform trachoma).

The third stage—trachoma III (Fig. 47*c*)—presents further progress in scar formation with residual infiltration of the submucous tissue and a small number of follicles. This period is especially prolonged, with possible aggravations of the disease and complications in the eyelids, the lacrimal passages and the cornea.

The fourth stage—trachoma IV (Fig. 47*d*)—is characterized by scarring. Scars like whitish streaks are found at first on the palpebral folds, and later on the conjunctiva of the tarsal cartilage, especially in its groove at the ciliary margin of the eyelid (sulcus subtarsalis). They may be delicate or rough, the latter causing contraction of the conjunctiva and often the formation of adhesions between the conjunctiva of the eyelids and that of the eyeball. Trachoma at this stage may be practically regarded as healed.

In the past, trachoma was the largest single cause of blindness owing to the serious complications it is likely to produce.

Complications and sequels of trachoma are noted *in the eyelids, the cornea, the conjunctiva and the lacrimal organs*, and are liable to produce severe lesions.

Cicatricial contraction of the conjunctiva and the tarsal cartilage, which becomes trough-shaped, produces inversion of the eyelid

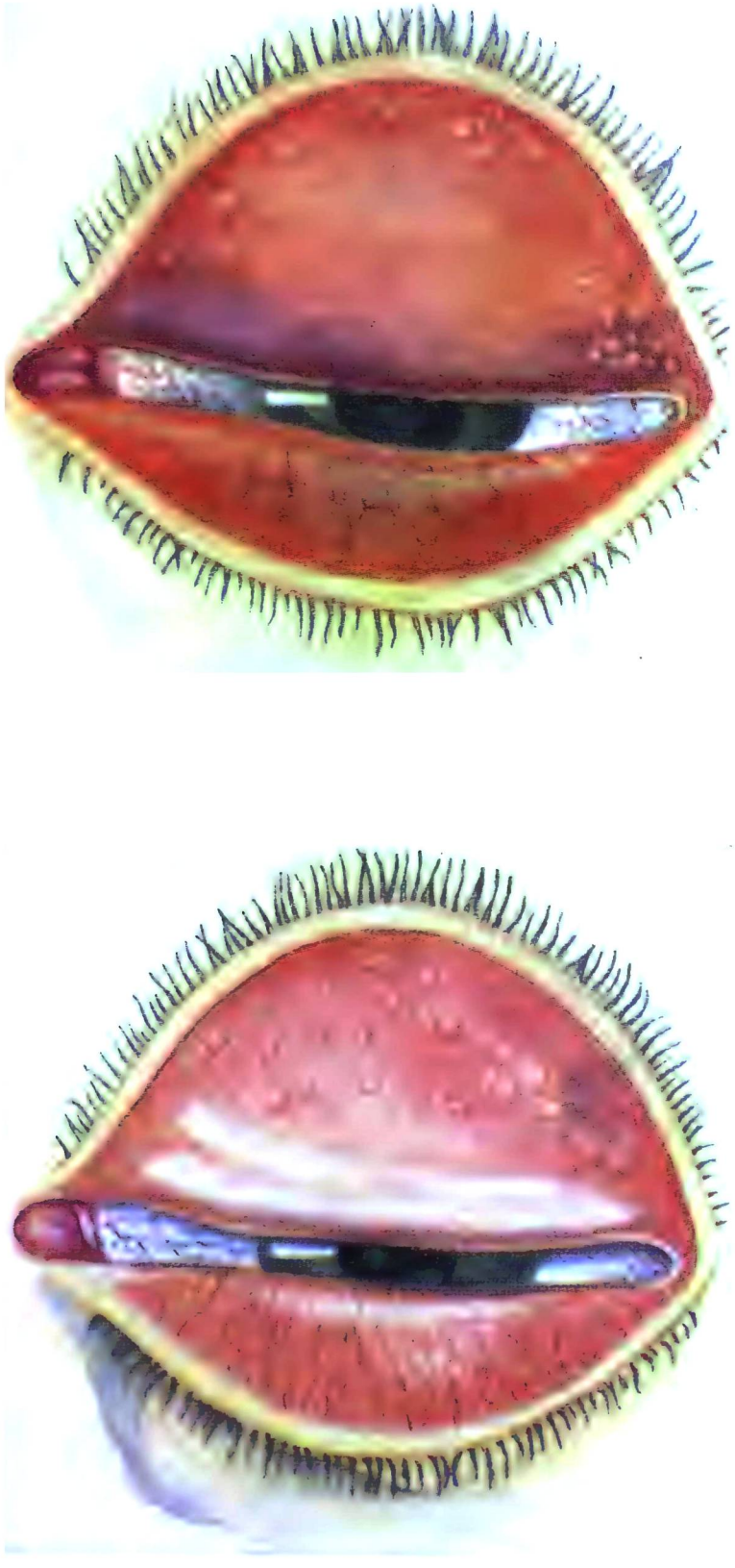

Fig. 47. Four stages of trachoma

a —trachoma I; *b* –trachoma II

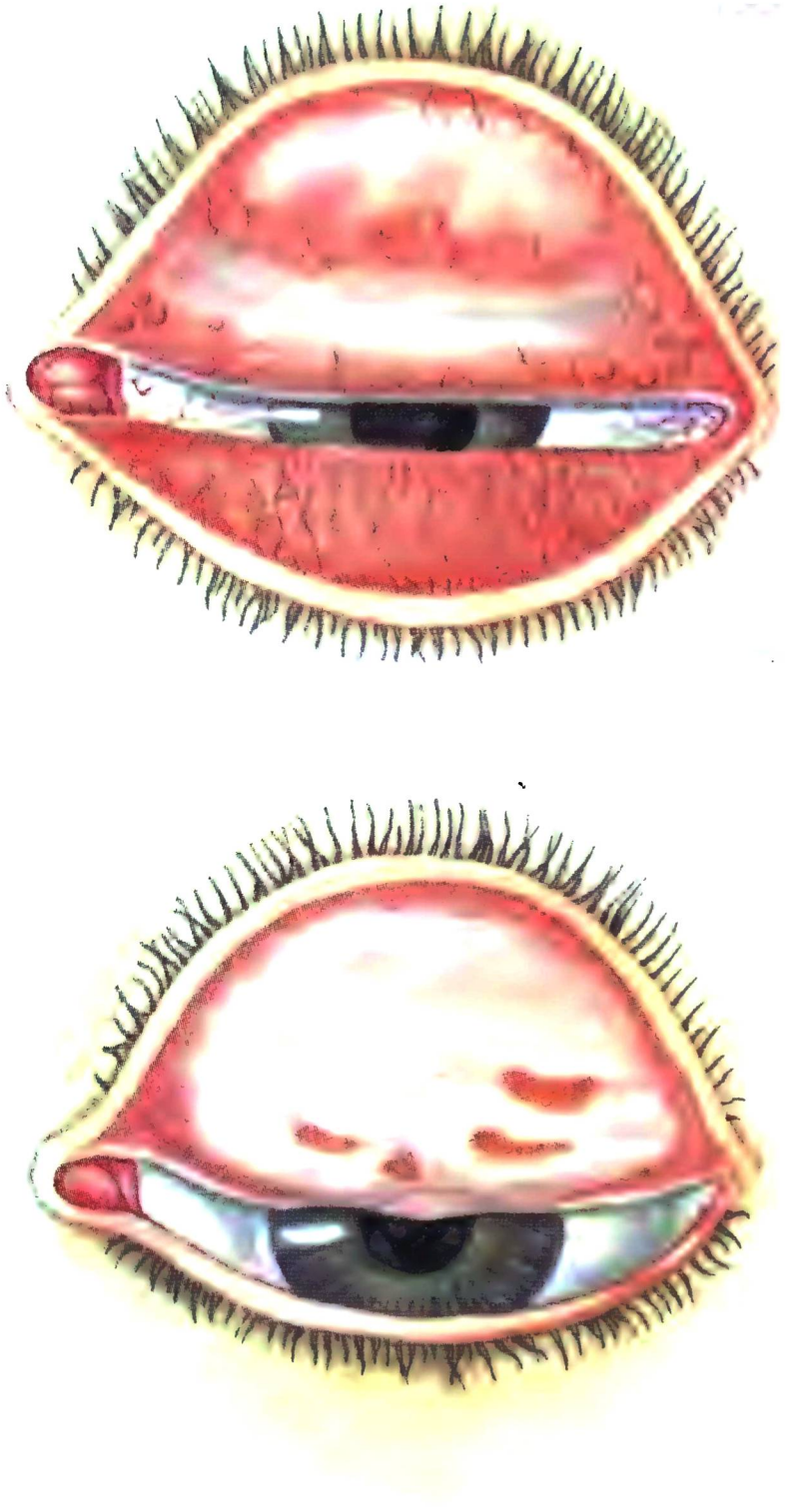

Fig 47. Four stages of trachoma

c—trachoma III; *d*—trachoma IV

(entropion). Scarring in the region of the hair-follicles of the cilia leads to contraction of the intermarginal space, obliteration of the posterior limbus of the palpebral margin and an abnormal position of the eyelashes, which is known as *trichiasis* (Fig. 48). The friction between the eyelashes and the eyeball causes irritation and ulceration of the cornea likely to produce opacity. Cicatricial contraction of the conjunctiva causes destruction of its glands and obliteration of the outlet ducts of the lacrimal gland. This process disturbs the moistening of the eyeball and causes dryness of the conjunctiva and cornea—*xerosis*. Derangement of trophic innervation also plays an important role in this process.

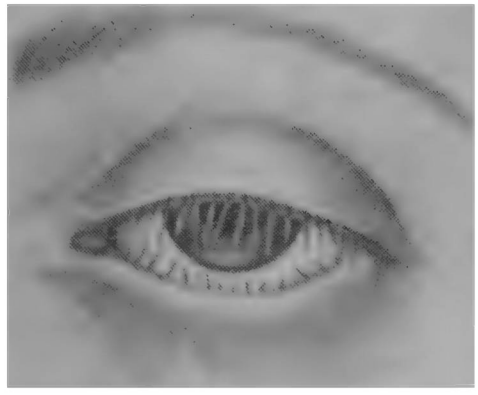

Fig. 48. Entropion (inversion) and trichiasis of the lid

Affection of the cornea by a superficial inflammatory vascularization known as *pannus* (pannus trachomatosus) is most severe in its upper half (Fig. 49). At first isolated superficial infiltrations of a grey colour form in the cornea, which undergoes vascularization with blood vessels extending from the conjunctiva. The infiltrations coalesce to form a fine opacity which ends in an irregular line along the border with the healthy cornea. This opacity of the cornea is a trachomatous infiltration analogous to that found in the conjunctiva.

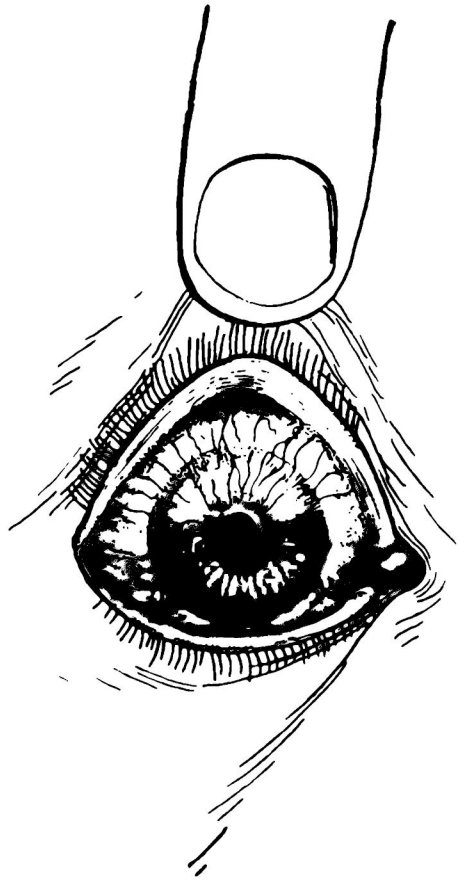

Fig. 49. Trachomatous pannus

According to the intensity of infiltration and the degree of vascularization of the cornea, pannus may be of a *delicate*, *vascular*, or *fleshy* character. In view of the invariable presence of pannus in all the stages of trachoma, includ-

ing the earliest one it has recently been suggested that this condition is not a complication but an inevitable symptom in trachoma as it extends from the conjunctiva to the cornea. Pannus leaves scars on the cornea and causes its opacity and sometimes, mostly in children, its extension and deformity (keratoconus). Deformity and opacity of the cornea result in a marked impairment of visual acuity.

Trachoma may be complicated by corneal ulcers forming at the border or outside the pannus, in the centre of the cornea. These ulcers are due to a secondary infection (with pneumococci, Koch-Week's bacilli) and are liable to cause leukoma (wall-eye) with all its consequences.

Complications in the conjunctiva are manifested by the trachomatous process being joined by acute conjunctivitis of any aetiology. If the latter precedes trachoma, it facilitates the entry of trachoma infection. If it joins trachoma already in existence, it aggravates its course, and contributes to complications and the spread of trachoma. Trachoma attended by conjunctivitis is especially contagious. That is why early diagnosis of conjunctivitis and vigorous therapy are highly important for the prevention of trachoma.

Complications in the lacrimal organs are manifested by inflammations of the lacrimal passages, which may be so severe as to cause chronic dacryocystitis. This is attended by continuous bleeding and pus discharge. The lacrimal puncta are dilated, the canaliculi sometimes swell under the skin, and pus is discharged into the conjunctival sac when pressure is applied to them. Disease of the lacrimal passages sustains the inflammatory process, contributes to exacerbations and a severe course of pannus, and to the formation of corneal ulcers.

DIFFERENTIAL DIAGNOSIS BETWEEN TRACHOMA, FOLLICULOSIS AND FOLLICULAR CONJUNCTIVITIS

The diagnosis of trachoma is based primarily on a thorough examination of the conjunctiva, above all its palpebral fold, and of the cornea. It is not difficult when the disease is manifest, especially after the formation of scars. In the early stage of the disease, however, when its symptoms are vague, it is sometimes difficult to make a diagnosis immediately, and trachoma has to be differentiated from such diseases as *folliculosis* and *follicular conjunctivitis*.

Folliculosis of the conjunctiva is characterized by the appearance of small superficial follicles with transparent contents (Fig. 50) arranged in regular rows on the unchanged mucosa of the palpebral

fold, chiefly of the lower eyelid. The disease ends with full resolution of the follicles, leaving no scars. The signs distinguishing folliculosis from trachoma are the absence of infiltration, scarring and complications characteristic of the latter disease, as well as the aspect of the follicles and the preferential localization of the morbid process in the region of the inferior palpebral fold (as opposed to trachoma).

Fig. 50. Folliculosis of the conjunctiva

Follicular conjunctivitis or *catarrh* is manifested by hyperaemia of the conjunctiva with a slight infiltration of the submucous tissue; larger follicles are arranged in rows, mainly in the region of the inferior palpebral fold (Fig. 51); sometimes they are scattered all over the conjunctival surface. The discharge is mucous or mucopurulent. There are no complications such as occur in trachoma, nor are any scars left upon recovery. It is sometimes difficult to differentiate between follicular conjunctivitis and trachoma immediately, and a certain period of observation is required. Such difficulties may crop up during examination of children of school age as a result of developmental changes in the conjunctiva—rapid growth of adenoid tissue accompanied by eruption of follicles on the palpebral conjunctiva. This is noted mainly in children with bronchoadenitis, scrofulosis, helminthiasis, and vitamin deficiency, as well as after acute conjunctivitis, when sanitary and hygienic conditions are poor, and in cases of eye strain because of inadequate lighting for lessons.

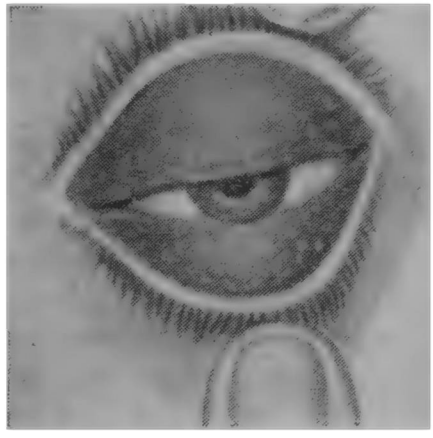

Fig. 51. Follicular conjunctivitis

The development of follicular conjunctivitis is facilitated by accompanying infections (Koch-Week's bacilli and other microbes, adenoviruses). To make a distinction between trachoma and follicular conjunctivitis the table drawn up by Prof. V. V. Chirkovsky can be used.

If a diagnosis is difficult to make, account should be taken of epidemiological evidence, such as the incidence of trachoma in a given area, at children's institutions, hostels, and in families, and of any other possibilities of contact with trachoma patients. If

such data are unavailable, in doubtful cases it is wise to avoid immediate trachoma therapy, which is likely to aggravate the disease; it should also be borne in mind that scars left on the conjunctiva by expressing granulations too roughly may prompt an erroneous diagnosis of trachoma.

No.	Follicular conjunctivitis	No.	Trachoma
1.	Prevailing affection of the conjunctiva of the inferior palpebral folds and usually slight affection of the superior palpebral folds	1.	Prevailing affection of the superior palpebral folds of the tarsal conjunctiva
2.	Infiltration, insignificant swelling of the conjunctiva	2.	Marked diffuse swelling and corrugation of the conjunctiva, multiple follicles
3.	Proneness of the follicles to form more or less regular rows in the region of the palpebral folds; the follicles are large, fleshly, pink-coloured elevations on the conjunctival surface	3.	The follicles are deeply implanted in tissue, are dull-grey in colour and have an irregular arrangement
4.	The cornea is not involved	4.	The cornea is affected very often with pannus or avascular keratitis
5.	No cicatricial changes in the cornea	5.	Scarring of the conjunctiva is the usual outcome

Therefore, when it is impossible to differentiate accurately between follicular conjunctivitis and trachoma, the treatment given during the initial period of four to five days is like that for acute conjunctivitis. Elimination of conjunctivitis symptoms makes differential diagnosis easier. If follicular conjunctivitis is diagnosed, the patient is given general therapy: intravenously or calcium chloride per os, vitamin therapy (above all vitamins A, B_2, C), and a diet with a limited carbohydrate content. Measures should be taken against helminthic invasions and specific therapy should be applied in cases of tuberculous bronchoadenitis. Correction of refraction anomalies and improvement of the patient's sanitary and hygienic conditions are also indispensable. Local therapy in-

volves synthomycin or penicillin solutions, or weak solutions of zinc sulphate with an addition of one drop of adrenalin per ml of solution. If the above therapy is continued for two to four weeks there is usually a marked improvement in the condition of the conjunctiva, a decrease in the number of follicles, and disappearance of infiltration, which confirms the diagnosis of follicular conjunctivitis.

In the scarring stage trachoma should be differentiated from changes caused by diphtheria and burns of the conjunctiva. It should be remembered that scars due to burns are rougher on the conjunctiva of the lower eyelid, the inferior palpebral fold and the cornea than those found in the region of the superior palpebral fold. Diphtheria leaves rougher scars on the eyelid conjunctiva than on the palpebral fold, and more often. Diphtheria is commonly confined to one eye, whereas trachoma affects both.

THE AETIOLOGY AND PREVALENCE OF TRACHOMA

Despite numerous efforts to identify its causal agent, the aetiology of trachoma remained obscure for a long time. Until 1907, trachoma was believed to be due to various micro-organisms discoverable in the conjunctival discharge. In 1907, Prowazek and Halberstedter discovered inclusion bodies near the cell nuclei in scrapings from the conjunctival epithelium. The role of these formations in the aetiology of trachoma was debated for more than forty years. Today, the virus theory of the origin of trachoma has been accepted by all. It has been found that the intracellular inclusions bear a relation to a virus.

Trachoma is a contagious disease conveyed from the sick to the healthy via objects in general use contaminated with particles of conjunctival discharge, such as a towel, a handkerchief, a pillow, a wash basin, or the hands of sick persons. The common fly is an important vehicle in the spread of trachoma; it may convey particles of purulent discharge on its legs from trachomatous eyes to the eyes of healthy persons. It is evident, therefore, that a trachoma patient is a permanent reservoir of infection, particularly in the family. Transmission of trachoma is favoured by inadequate sanitary and hygienic conditions, overcrowded apartments, a low level of culture, and inadequacy or absence of medical services. It is evident that trachoma is a disease rooted in social conditions, and its prophylaxis and therapy can be successful only after the factors mentioned above have been eliminated.

Proof of this is the fact that in tzarist Russia trachoma was widespread all over the country, especially in the most backward territories, such as Central Asia, among the Chuvashs, the Yakuts, etc.

In the Kazan Province, for example, according to records of the all Russia census of the blind, trachoma was found in 25 per cent of the population in 1897, and ranked first among the causes of blindness (21.4 per cent). The percentages of trachoma and blindness due to it were highest in the rural areas. After the Great October Socialist Revolution a campaign was launched to eradicate trachoma. Institutes, clinico-prophylactical centres (dispensaries) and medical stations for trachoma control were set up, and medical personnel were given wide public support in these activities.

Today trachoma patients are found only in isolated localities, and conditions are favourable for the early and complete eradication of this disease in the Soviet Union. The achievements of Soviet health services are especially notable when viewed against the background of trachoma incidence in the world, mainly in the underdeveloped countries where there are 400 million trachoma patients.

ORGANIZATION OF TRACHOMA CONTROL

One of the basic measures for eradicating trachoma is to check the spread of fresh cases of infection. This can be attained only by early diagnosis and therapy of all active cases and by high standards of public hygiene.

The growing welfare and cultural standards of the population, the wide scope of clinico-prophylactic measures, constant improvement of the methods of therapy, and the introduction of new and more effective remedies together with health education are the basis for prophylaxis and the effective control of trachoma. The personnel of rural medical centres play the leading role in trachoma control. They carry out complex measures to detect and treat trachoma cases in particular areas. The personnel of the medical stations and the medical and obstetric centres are briefed on trachoma at special seminars directed by an ophthalmologist, before they undertake an overall examination of the population. When a case is detected it is shown to the district physician and then to an ophthalmologist; if the diagnosis is confirmed the case is registered on a special card. Such a card is provided for every new applicant for medical aid in the current year no matter whether or not he has been put on record in past years.

The card indicates the stage of the disease so that changes in the sickness rate can be registered. For example, cases of the highly contagious stages of trachoma (I-II) point to the need for vigorous therapy and sometimes for solution of a trachoma focus and quarantine. Conversely, the prevalence of cases of trachoma III is evidence of the effectiveness of measures for trachoma control and indicate that prospects for its eradication in the given area are good.

At all medical institutions, including medical and obstetric centres and trachoma control posts, every trachoma case is recorded in a special out-patients card indicating the diagnosis (always with reference to its stage), regularity of therapy, and the effectiveness of the measures taken.

Prophylactic examination of the population, especially of schoolchildren, pre-conscripts, and members of the families of trachoma patients, should be carried out regularly, at least twice a year.

Medical workers must make vigorous efforts to help raise the cultural and sanitary standards of the people, and persuade them to take measures for individual prophylaxis. In view of the epidemiological evidence available on the ways in which trachoma spreads special attention should be given to personal hygiene in families with trachoma patients. Another highly important measure is fly control.

Each day medical personnel must carry out persistent work in health education to make the public aware of the hazards of trachoma, to vision in particular, and of the ways in which it is transmitted.

They should help the population acquire sanitary and hygienic habits and understand the need for early medical aid, regular therapy until full recovery and subsequent observation.

Instruction by way of health education is given during the examination of patients, in consulting hours, in discussions with local residents, at school lessons, and by means of posters, booklets, films, etc. The work is carried out under the control and guidance of the physician in charge of the rural medical centre, together with the ophthalmologist, in accordance with the programme they draw up. Besides the treatment given at the out-patient centre, medical personnel visit patients at home, examine members of their families to detect fresh trachoma cases, and inspect their living conditions. If all members of a family are well, they are put under constant observation so as to find out early forms of trachoma likely to occur during contact between sick and healthy persons.

Consequently, the basic measure in trachoma control is to bring clinico-prophylactic assistance within the reach of all. This assistance is based on the dispensary method, i.e., on early detection and accurate registration of trachoma cases, treatment for all without exception, prophylactic examination of the population and health education.

TREATMENT OF TRACHOMA

The following scheme for treating trachoma with tetracycline-series antibiotics—biomycin (chlortetracycline), terramycin, tetracycline—has been recommended recently according to the metho-

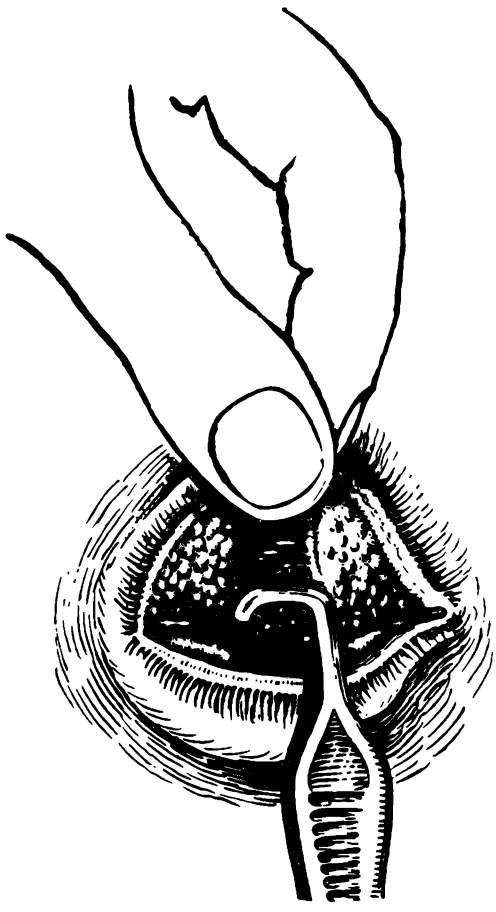

Fig. 52. Expression of trachoma granulations

dologic instructions drawn up by the Helmholtz State Scientific Research Institute of Eye Diseases and is approved by the USSR Ministry of Public Health (1960). Treatment is begun by applying these antibiotics in ointment form to the conjunctival sac five times a day and is continued for two or three weeks. This is followed by careful expression of the remaining follicles. It is usually enough to express the follicles once during a course of treatment. If isolated follicles are found at a later time, they are destroyed with small trachoma forceps following anaesthesia of the conjunctiva with a 0.25 per cent dicaine solution. After expression, ointment is again applied five times a day. Expression of the follicles should not be undertaken before or in the early days of drug therapy since this is likely to exacerbate the trachomatous process. On the contrary, expression after antibiotic therapy usually causes no aggravations, and leaves delicate scars on the conjunctiva. Expression is undertaken if follicles are really present, and is contraindicated for infiltration of the submucous tissue alone and for the papillary form of trachoma. It is also avoided in cases of purulent discharge, exacerbation of pannus and ulceration of the cornea.

The procedure of expression is as follows: the skin of the eyelid is wiped with alcohol; following the triple drop instillation of a 0.5 per cent dicaine solution, a subconjunctival injection into the palpebral folds of a 1 per cent dicaine solution or a 1 per cent novocain (procaine) solution is given; the eyelids are everted, and the conjunctiva is seized with the jaws of a trachoma forceps (Fig. 52). Follicles are expressed all over the surface of both eyelids; the manipulations are easier to perform with the patient lying down. When the follicles have been expressed the conjunctival sac is irrigated with mercury cyanide or synthomycin solution followed by the application of one of the above preparations. Cold eye lotions are applied to the eye to relieve pain and reduce oedema of tissues.

On a doctor's instructions, the medical assistant may undertake expression of follicles himself. During this operation, medical personnel should wear glasses or a special protective mask to avoid the risk of infection with trachoma from particles of the contents of the trachoma granulations.

The most stubborn stage of trachoma is the third stage in which the best curative effect is produced by a 1 per cent dibiomycin ointment applied once a day to the conjunctival sac. To prevent relapses, the same ointment is used for six months for the fourth stage of trachoma. A patient recognized by an ophthalmologist as cured is removed from the sick record. Those who have recovered from trachoma, however, are kept on dispensary records for two to three years and are examined by an ophthalmologist once every three months.

In the majority of cases, treatment lasts from two to three months, but sometimes it takes longer.

Treatment is given with due attention to the patient's general condition. Stubborn trachoma resisting therapy is usually found in conjunction with scrofulosis, tuberculosis, malaria, and syphilis, as well as with helminthiasis, particularly in children. In such cases, treatment is directed towards improving the general health. The following are found to be very helpful: calcium chloride, vitamins, autohaemotherapy, general health promotion and specific therapy (against tuberculosis, syphilis, malaria, etc.).

In especially severe cases of trachoma, severe lesions of the cornea, and failure of local therapy, the latter is supplemented with peroral administration of sulpha drugs or tetracycline-series antibiotics: in adults a 0.25 g dose is given 4-5 times a day for a period of 7-8 days, while in children a daily dose of 0.025 g per kg of body weight is given. Should one course of treatment prove inadequate, it is repeated 2-3 weeks later. Sometimes these drugs cause side effects, such as nausea, vomiting, stomatitis and dermatitis. In such cases, the drug is given in a smaller dosage or is withdrawn for a time; sometimes, the method of therapy has to be changed.

In stubborn forms of trachoma, other drugs should be used after 3-4 months of treatment; suspension of local therapy 2-3 months after it is begun may prove helpful. There should also be an interval in therapy when there are symptoms of irritation of the conjunctiva.

Patients with relapsing forms of the disease, complicated with pannus, xerosis, entropion and trichiasis are admitted to an eye clinic for therapy and, when required, surgery. The latter is inevitable in cases of entropion and trichiasis for correcting the position of the eyelids.

10

DISEASES OF THE CORNEA AND SCLERA

DISEASES OF THE CORNEA

Because of its exposed position the cornea is most vulnerable to external influences, and is often injured. Its close anatomical relationships with the conjunctiva, sclera and choroid are often responsible for their being simultaneously involved in many systemic diseases (syphilis, tuberculosis).

Diseases of the cornea are regarded as serious because both in the acute period and later the cornea becomes less transparent, its form changes, and the acuity of vision becomes impaired. As a result, permanent opacity may develop, ranging from translucency (haziness) to a rough scar, known as the wall-eye, which is a frequent cause of total blindness.

The cornea has a slightly irregular spherical shape. It is shiny like a mirror, transparent and highly sensitive to the touch. The disturbance of these properties accounts for the variety of clinical symptoms noted in corneal diseases. Pathological changes in the cornea are revealed by visual examination in daylight, examination in a dark room with lateral or focal lighting through a binocular lens, study of corneal transparency in transmitted light, and by the light of a slit lamp.

To detect a defect on the corneal surface, a 2 per cent fluorescein-sodium solution is introduced into the conjunctival sac, and excess is washed off with water or physiological salt solution. During this procedure an abrasion on the epithelium takes on a greenish colour. The corneal sensation is examined by touching the cornea with several cotton-wool fibres, which normally causes a reflex closure of the eyelids. Examination of the cornea should be followed by a careful inspection of the entire eyeball, bearing in mind that the iris is especially liable to be involved in the disease. A thorough general examination of the patient is also required so as to identify the disease aetiology.

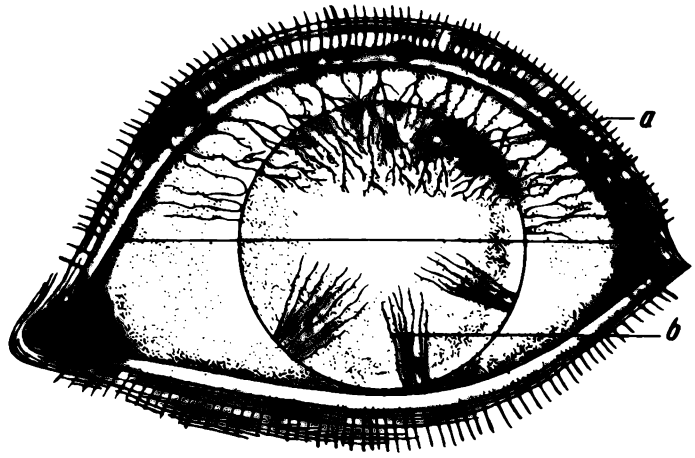

Fig. 53. Ingrowth of vessels into the cornea
a—intercommunicating superficial vessels form a bright-red network; *b*—deep vessels form separate sheaves

The most frequent disease of the cornea is *keratitis*, an inflammation of exogenous or endogenous origin. Among the aetiological factors of exogenous keratitis, mention should be made of acute conjunctivitis, dacryocystitis, injuries, etc., whereas the development of endogenous keratitis is favoured by tuberculosis, syphilis, virus infections, derangements of trophic innervation, etc.

Depending on the depth of location of the inflammatory focus keratitis may be superficial or deep (interstitial) and give different clinical symptoms. The causes of superficial keratites are usually associated with exogenous factors, and interstitial keratites with endogenous ones.

General signs of keratitis. Inflammation of the cornea is attended by symptoms of irritation of the eye: palpebral spasm, lacrimation, photophobia, and pain. All these symptoms are more manifest in superficial keratites because the sensory endings of the trigeminal nerve are more involved. As a rule, superficial keratitis is accompanied by superficial or mixed hyperaemia, injection of the conjunctival vessels and their growth into the cornea, whereas in interstitial keratitis the same changes are observed in deep-lying tissues (Fig. 53).

These vessels differ in appearance: the superficial ones communicate widely with one another, forming a network of a bright-red colour; deeper vessels become visible only at the limbus, grow into the cornea in the form of panicles and are not connected. Keratitis produces an inflammatory infiltration which is manifested by haziness of the cornea, and impairment of its normal lustre. In superficial keratitis, defects of the epithelium are found often, whereas in interstitial keratitis the cornea retains its brilliance and mirror-like lustre and no defect forms in the epithelium.

In both forms of the inflammation the haziness of the cornea is greyish in colour and there are no distinct boundaries with the surrounding tissue owing to inflammatory oedema around the hazy parts. This is a distinguishing sign of keratitis, i.e., an inflammation, from the resulting white coloured scar with distinct outlines, the other signs of inflammation being absent. Very often keratitis is accompanied by inflammatory symptoms in the iris manifested by its changed colour and relief and by contraction of the pupil. Exudate and pus frequently accumulate in the anterior chamber of the eye. Lumps of exudate deposited on the posterior surface of the cornea in interstitial keratitis are called precipitates.

Keratitis may take an acute or chronic course and often relapses.

Superficial Keratitis

This disease is characterized by small grey elevations—infiltrates—appearing in the superficial layers of the cornea, usually on the border with the limbus. Coalescence and nonpurulent ulceration are likely, producing a confined area of fine haziness. If there is no ulceration appropriate treatment may bring about complete resorption of the infiltrates, the cornea retaining its transparency.

Superficial keratites occur as complications of acute conjunctivitis and dacryocystitis. The connection between keratitis and affection of the meibomian glands can easily be found from hyperaemia of the margins of the eyelids and from the fatty secretion expressed from the efferent ducts of the glands during massage of the margins of the eyelids (see Fig. 30).

Treatment of superficial keratitis is aimed at eliminating its cause. In addition, symptoms of concomitant iritis are treated by instilling atropine or scopolamine solutions into the eye. The presence of iritis indicates that the disease is serious, and the patient should be sent to an ophthalmologist as early as possible.

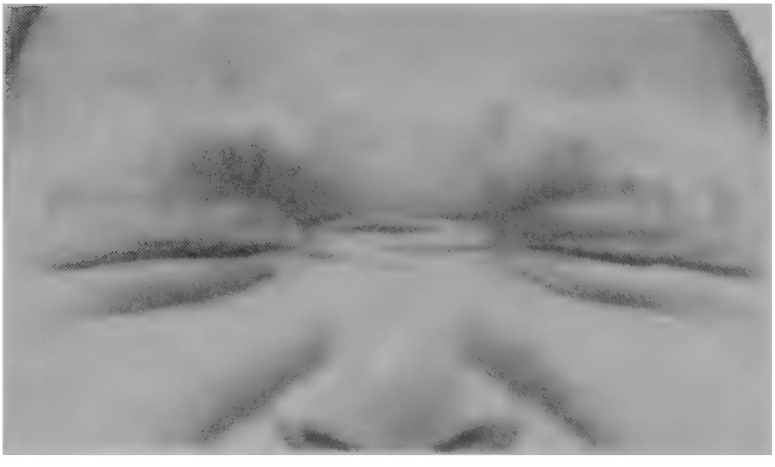

Fig. 54. Palpebral spasm in scrofulosis

Superficial keratitis may be due to superficial lesions of the cornea, especially from wedged-in foreign bodies, or to a gaping palpebral fissure (*lagophthalmos*). Once detected, a foreign body should be extracted. Treatment for lagophthalmos is the same as for traumatic keratitis.

Scrofulous Keratitis

Scrofulous keratitis is a tuberculous-allergic inflammation of the cornea, and is encountered mainly in children with bronchoadenitis or tuberculosis of the lymph nodes. Its development is favoured by weakened body resistance after influenza, measles, whooping-cough, and chicken-pox. The disease is chronic, with numerous relapses. The course of scrofulosis is aggravated by helminthic invasions, vitamin insufficiency, a diet rich in carbohydrates (pastries, sweetmeats), and poor sanitary and hygienic conditions. The principal signs of scrofulous keratitis are intense photophobia, palpebral spasm, and lacrimation (Fig. 54). Eczematous weeping rashes often appear on the facial skin, particularly at the wings of the nose, as well as on the scalp and behind the auricles. The face, especially the nose and lips, and the submaxillary lymph nodes are swollen. When the palpebral fissure is opened after being compressed by a spasm there is a profuse discharge of tears collected in the conjunctival sac. Because of photophobia, examination of the child's eyes is difficult. His head has to be held firmly between the examiner's knees (see Fig. 21) and the eyelids are drawn apart by means of palpebral levators. The eyeball conjunctiva at the limbus (Fig. 55) or the cornea itself (Fig. 56)

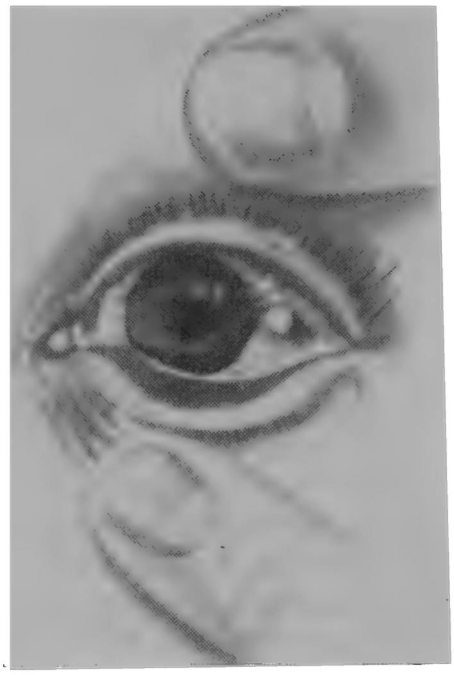

Fig. 55. Phlyctena of the conjunctiva

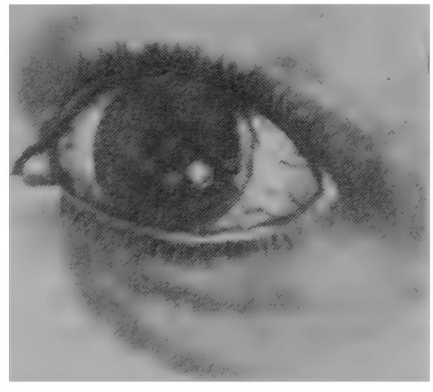

Fig. 56. Phlyctena of the cornea

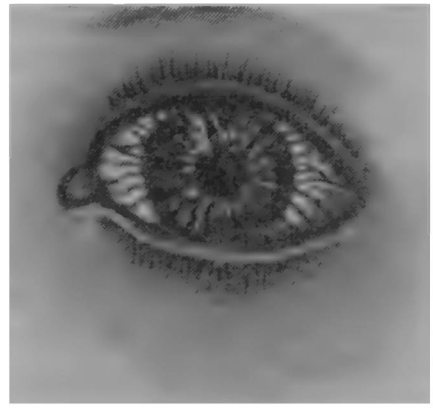

Fig. 57. Scrofulous pannus

is covered by *phlyctens*, small transparent nodules made up of lymphocytes, epithelioid and giant cells, and accompanied by a bundle of conjunctival vessels.

Phlyctens may be *large*, *isolated* (*solitary*) or *small*, *multiple* (*miliary*). The course of the disease is variable: the nodules may resolve, leaving only hazy dots or they may extend to the centre of the cornea along with the bundle of vessels (fascicular keratitis). Disintegration of tissue is likely to produce a central or peripheral ulcer of the cornea. A deep-set ulcer on the periphery of the cornea may cause perforation of the latter and prolapse of the iris, which results in a wall-eye adhering to the iris.

This disturbs intra-ocular fluid drainage which frequently leads to an increase in the intra-ocular tension (secondary glaucoma) which is injurious to vision. The eye may also be destroyed if perforation of the ulcer is followed by secondary infection invading the internal tissues of the eyeball (endophthalmitis).

In severe cases of scrofulous keratitis, the entire cornea is pierced by superficial vessels. This condition is known as *scrofulous pannus* (Fig. 57), which, unlike trachomatous pannus, may begin in any part of the limbus and is usually circular.

Treatment. Both general and local measures are applied. First of all, a 5 per cent calcium chloride solution in a dose of one tea- or tablespoonful (depending on the child's age) is given per os three times a day. Intramuscular injections of a 0.25 per cent calcium chloride solution are even more beneficial. One spoonful of cod-liver oil must be taken three times a day (except in the warm season of the year); riboflavin with ascorbic acid taken per os also aids recovery. It is helpful to have a diet low in carbohydrates, especially in sugar and sweets. Phthivaside and PAS are given per os, and streptomycin by intramuscular injection (as prescribed by the phthisiologist). It is desirable to give the child total irradiation with ultraviolet light (with permission from the pediatrician or phthisiologist). Sanitary and hygienic conditions should be improved and the patient should stay in the open air for a long time preferably in a forest.

Local treatment involves instilling into the eye streptomycin drops 4-5 times a day, adrenalin, or cortisone suspension, and applying penicillin or yellow mercury ointment to the eyelid conjunctiva.

In cases of contraction of the pupil and marked symptoms of iritis, the pupil is dilated with atropine solution (two or three times a day, depending on its effect). Atropine is contraindicated for deep corneal ulcers situated near the limbus and liable to perforation. In such cases, eserine or pilocarpine is used.

Fissures of the skin near the angles of the palpebral fissure are cauterized with a 10 per cent silver nitrate solution. At the regres-

sive stage of the disease, resolution of opacity is favoured by prolonged application of yellow mercury ointment and dionine solutions in increasing concentrations.

The patient should be examined at a tuberculosis control centre and should be kept under observation both by an ophthalmologist and by a phthisiologist.

Neurogenous or Neurotrophic Keratites

Neurogenous or *neurotrophic* keratites are caused by various infective agents, particularly by viruses (herpes simplex, herpes zoster, adenoviruses), and may also be due to compression of or injury to the trigeminal nerve or its ganglia. Irrespective of their causes, neurogenous keratites are characterized by connection with a lesion of the trigeminal nerve, a sharp reduction or absence of the corneal sensation, slow reparation of corneal defects, a protracted course, and liability to relapse.

Herpetic Keratitis

Herpetic keratitis may also occur with malaria, diseases of the teeth and paranasal sinuses. This form of keratitis is accompanied by palpebral spasm, lacrimation and photophobia, pains in the eye and the head.

Herpetic keratitis is known to occur in two forms: herpes febrilis and herpes zoster (shingles).

Herpes febrilis is characterized by the appearance under the epithelium of vesicles containing fluid, and causing detachment of the epithelium. Later, the vesicles break down leaving epithelial defects and erosions, which coalesce with one another acquiring peculiar shapes. They are superceded by infiltrations, dendritic keratitis (Fig. 58) and, after a protracted, torpid and recidivating course, leave stable opacities of the cornea.

Herpes zoster causes the eruption of vesicles on the skin of the forehead, eyelid and cheek, following the course of the first branch of the trigeminal nerve. Such vesicles are often seen on the cornea also. The disease is accompanied by sharp pain. The vesicles leave scars on the skin.

Treatment is prescribed according to the aetiology and course of the disease; in virus infection, synthomycin per os in doses of 0.5 g six times a day for a period of five to seven days, and daily intravenous infusions of 10-20 ml of a 40 per cent glucose solution with a 5 per cent thiamine solution and a 5 per cent ascorbic acid solution (in 1 ml doses).

In herpetic keratitis of any aetiology, riboflavin with ascorbic acid and tissue therapy are very beneficial. Local treatment in-

Fig. 58. Dendritic keratitis

volves instillation into the eye of atropine and quinine, and the application of ointments, preferably antibiotics. Novocain blocking is very effective; the drug is injected subconjunctivally in a 2 ml dose every other day, or into the muscular tissue of the eyelid in a 5 ml dose, or retrobulbarly in a 2 ml dose. The disease requires the attention of a neuropathologist also.

Epidemic Keratoconjunctivitis of Adenovirus Aetiology

This disease sets in just like simple conjunctivitis, with the eruption of follicles (usually on the conjunctiva of the lower eyelid). Within seven to ten days, delicate punctate or coin-shaped superficial infiltrations appear on the cornea, which loses its sensation. In certain cases, these signs are joined by iridocyclitis which causes contraction of the pupil and deposition of precipitates on the posterior surface of the cornea. The disease develops at different times after catarrh of the upper air passages or a superficial injury to the eye, especially from small wedged-in foreign bodies, and after bathing in stagnant water. The disease is highly contagious, and is passed on through objects in general use, by the hands of medical personnel, through tonometers, medicine-droppers, water in public swimming-pools, and in bath-houses. Prophylaxis involves quarantine and therapy, washing of the hands by medical personnel after handling a patient, thorough sterilization of instruments, tonometers, medicine-droppers, etc.

Treatment. Ethasole and calcium gluconate are given per os for seven to ten days; levomycetin ointment, cortisone, and atropine are applied locally for signs of iritis. Treatment is carried out by an ophthalmologist.

Creeping Ulcer of the Cornea (Ulcus Serpens)

Creeping or serpiginous ulcer is characterized by a corneal defect, usually central, with purulent infiltration, which is liable to spread over the surface or into the depth of the cornea. The ulcer is sickle-shaped, with an undermined edge raised above the surface and gradually creeping across the cornea (Fig. 59). At the same time, the ulcer heals on the opposite side. The bed of the ulcer is covered

with a dull purulent coat, and the cornea around the ulcer is hazy and oedematous. An accumulation of pus (hypopyon) is found on the floor of the anterior chamber of the eye. The iris is discoloured, the pupil is often irregular in form due to adhesions with the anterior capsule of the crystalline lens. These adhesions may interfere with drainage of intra-ocular fluid and cause secondary glaucoma. The disease of the cornea is attended by oedema and spasm of the eyelids, lacrimation, photophobia and a considerable loss of vision. An ulcer running a favourable course heals with cessation of purulent infiltration, epithelization of tissue defects and formation of corneal opacities varying in degree with the intensity of ulceration. If the course is unfavourable, the ulcer spreads over the surface or into the depth of the cornea, causing destruction and perforation of the cornea with extension of the suppurative process to the internal tissues of the eye (endophthalmitis) or to the entire eyeball and retrobulbar cellular tissue (panophthalmitis). The disease results in atrophy of the eye and blindness. The latter may also be caused by secondary glaucoma.

Creeping ulcer commonly occurs after superficial injuries to the cornea by ears of corn, straw, and tree branches during farm work, as well as by small foreign bodies. It is largely encouraged by the presence of purulent dacryocystitis which acts as a constant reservoir of infection in the eye. Another factor which undoubtedly

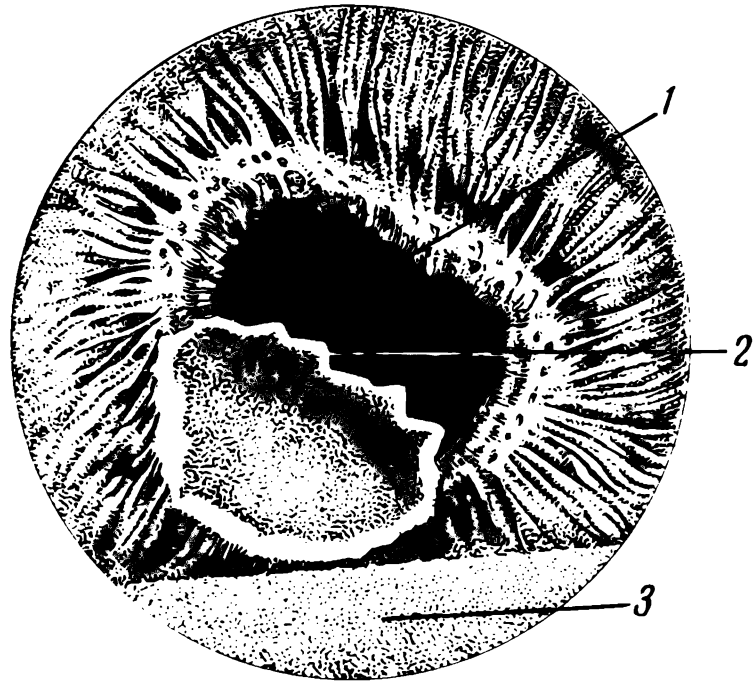

Fig. 59. Creeping ulcer of the cornea
1—posterior synechia; *2*—growing edge of the ulcer; *3*—hypopyon

contributes to the pathogenesis of creeping ulcer is low body resistance, and a partial or total loss of the corneal sensation. That is why it is more frequently encountered in senile persons, particularly those with various constitutional disorders.

Prophylaxis of creeping ulcer entails the early and careful extraction of foreign bodies from the cornea, followed by treatment for traumatic keratitis. When examining a patient with a corneal injury, special attention should be given to the condition of *the conjunctiva* and *the lacrimal passages.* If there is disturbance in tear drainage and especially if dacryocystitis is detected the patient should immediately be sent to an ophthalmologist for surgery. The latter is indicated also for a fresh corneal ulcer.

Treatment. Today, creeping ulcer of the cornea has lost its significance as a formidable disease thanks to successful general and local therapy with sulphonamides and antibiotics, particularly in the early stage of the disease. Good therapeutic effects are produced by penicillin solutions administered by drop instillation and subconjunctival injections, while the application of synthomycin or levomycetin ointment 4-5 times a day is especially beneficial. Synthomycin per os, intramuscular penicillin injections, vitamins A, C and B_2, tissue therapy and other types of general therapy are indicated for a grave course of ulcer resisting treatment.

If the intra-ocular tension has not increased, a patient is, in addition, given frequent instillations of atropine so as to cure the inflammation of the iris and prevent adhesions with the crystalline lens or cornea. In cases of glaucoma frequent instillations of pilocarpine are given, and should the intra-ocular tension fail to decrease, surgery is undertaken. The latter involves incision of the cornea within the limits of the healthy tissue or within the ulcer itself and excision of fistulas in the cornea, or therapeutical transplantation of the cornea, iridectomy for glaucoma, etc.

The operation helps to remove pus from the anterior chamber, improve humor metabolism, terminate the flux of painful impulses to the brain cortex and thereby improve the trophic supply of the cornea. Certain types of surgical interference, however, leave a rough wall-eye which markedly impairs the visual functions. This circumstance means that surgery must be restricted to exactly specified cases.

An even more severe disease known as *keratomalacia*, a purulent softening of both corneas, is encountered mainly in infants following grave emaciating diseases of the gastrointestinal tract.

Treatment. In addition to the above-described local therapy, blood transfusion, vitamin therapy (especially with vitamin A), and intramuscular penicillin injections are used. The outcome of the disease largely depends on the patient's general condition.

Deep or Interstitial Keratites

This form of disease produces a focal or diffuse infiltration of the deeper layers of the cornea without proneness to ulceration. This makes the complete resolution of opacities possible with early and adequate therapy. Otherwise, the infiltrated parts degenerate into connective tissue with a rough wall-eye which markedly impairs the visual acuity.

Interstitial keratites are associated with congenital syphilis, tuberculosis, malaria, brucellosis, and virus infection. The provoking factors are superficial and blunt injuries to the eye, previous infectious diseases, particularly influenza, and vitamin deficiency, as well as pregnancy and lactation in women.

Keratitis associated with congenital syphilis (Fig. 60) gives a characteristic clinical picture. It is commonly encountered in children of school age, first affecting one eye and later often extending to the other.

In a perfectly normal eye or in an eye with slight signs of irritation in the deeper layers of the cornea, usually on the border with the limbus, there appears a delicate diffuse infiltration, and the cornea presents a ground-glass appearance. The opacity gradually spreads towards the centre and the limbus on the opposite side. Already deep injection of the eyeball conjunctiva and, as a rule, signs of concomitant iritis can be seen. In certain cases, the cornea is pierced by deep vessels passing in the form of panicles (see Fig. 53) from the limbus to the centre.

Other signs of congenital syphilis are also found. These are impaired hearing, saddle-nose deformity, curved sabre-shaped legs, absence of the xiphoid process of the sternum, and widely spaced cone- or

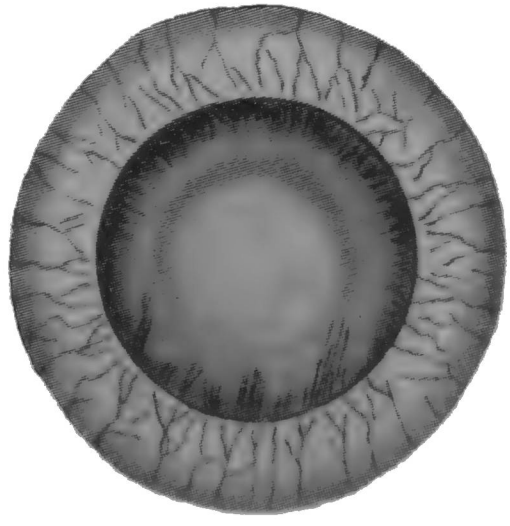

Fig. 60. *Deep keratitis associated with congenital syphilis*

Fig. 61. Hutchinson's teeth

peg-shaped teeth without enamel, with a semilunar notch in the incisor teeth (Hutchinson's teeth) (Fig. 61). Wassermann's test is often positive. The disease usually runs a protracted course, lasting for months. If adequate early treatment is given, keratitis may have a favourable outcome, and there may even be complete resolution of the corneal opacity. If the outcome is unfavourable, the disease may become complicated by iritis, causing adhesions along the pupillary margin, and by secondary glaucoma. This ultimately leads to a rough wall-eye and severe impairment of visual acuity, even to the extent of total blindness.

Treatment is both general and local. General therapy consists in a course of intramuscular injections of penicillin (at least 5,000,000 U), specific preparations prescribed by a venereologist, and measures to improve the general health. Local therapy involves heat (hot-water bottles, fomentation, compresses, sollux lamp), and atropine is also used. Subconjunctival injections of cortisone are very beneficial. Pilocarpine is used when the intra-ocular tension has increased; dionine is given at the same time in increasing concentrations to encourage resolution of the opacity. In the regressive period, dionine is combined with a prolonged application of yellow mercury ointment and electrophoresis with potassium iodide. Tissue therapy is highly effective in all the stages of the disease.

Deep Tuberculous Keratitis

This disease is encountered mainly in juveniles and young people, but it may affect older persons as well. Usually the disease attacks one eye first. It is prone to aggravations and relapses caused by various systemic diseases and unfavourable surroundings.

The disease is characterized by marked signs of eye irritation, confined infiltrations tending to coalesce in the corneal stroma, and an ingrowth of deeper and superficial vessels in the cornea (Fig. 62). The onset of keratitis is preceded by an inflammation of the uveal tract like a specific tuberculous metastasis, while the corneal involvement is a secondary process. A patient with tuberculous lesions of the eye is usually regarded by phthisiologists and roentgenologists as practically healthy, since the only discoverable signs are traces of the

primary complex of tuberculous infection in the bronchial glands (Ghon lesion), and the eye disease is often the only active tuberculous focus in the patient's body. In tuberculous keratitis the body temperature is subfebrile, rising chiefly in the daytime. The Mantoux test and the focal test—reaction to subcutaneous injection of tuberculin—are positive. A comprehensive examination helps a correct diagnosis to be made. Keratitis runs a protracted course, and is liable to exacerbate and relapse, especially after such conditions as influenza or a trauma. Upon recovery, tuberculous keratitis often leaves a permanent opacity with an ingrowth of blood vessels.

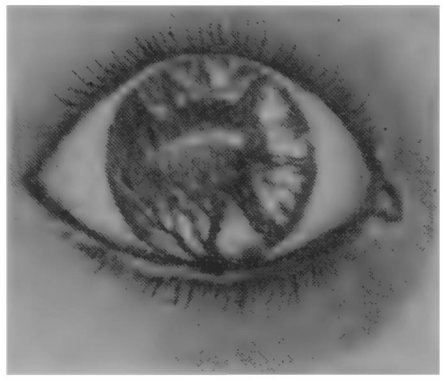

Fig. 62. Deep tuberculous keratitis

Treatment. General therapy is the same as for scrofulous keratitis, but it is often necessary to employ tuberculin therapy, which should be carried out at an eye hospital by an experienced ophthalmologist who is constantly in touch with a phthisiologist. It is very helpful to give vitamins A, B_2, C, and D_2 per os. A diet low in carbohydrates and salts, and climatic therapy at a health resort are also indicated. Local therapy uses atropine (3-4 times a day), streptomycin (25,000 U daily or every other day) which is administered in a 1 ml dose subconjunctivally or by means of electrophoresis. When the acute symptoms have abated the resolution of opacity is promoted with dionine, yellow mercury ointment and tissue preparations used carefully in small doses (0.2-0.3 ml per injection). The latter are also helpful for a new torpid process resisting therapy.

Keratites of Malarial and Brucellar Aetiology

Clinically these diseases are very much like tuberculous keratitis. In each case, their aetiology can be ascertained only by means of a clinical and laboratory examination of the patient. Local treatment is the same as for other types of keratitis; general therapy depends on the aetiology of the disease.

Healed keratitis leaves opacities of variable intensity and localization, depending on the character of the disease. Vision is impaired to a variable extent. For example, a fine haziness (nubecula), a more intense small confined opacity (macula), or an even more intense and larger opacity known as the wall-eye (leukoma) may develop.

The wall-eye may involve part of the cornea (*partial leukoma*) or its entire surface (*total leukoma*). The former may be *central* or *peripheral*. Ulceration of the cornea attended by perforation,

prolapse and incarceration of the iris produces *a wall-eye adherent to the iris* (leukoma adhaerens). Such a wall-eye interferes with the normal drainage of intra-ocular fluid and causes glaucoma. Bulging of the wall-eye alone (*ectasy*) or of the entire cornea (*staphyloma*) is likely to occur. Finally, *a flattened wall-eye* may also develop in cases where the intra-ocular tension has decreased. A wall-eye may have an extensive ingrowth of blood-vessels. This condition is known as *vascularization*.

The wall-eye causes loss of vision, to the extent of total blindness. The severity of this loss is directly related to the intensity of opacity and its proximity to the centre of the cornea. Increased intra-ocular tension is especially detrimental to vision; surgery should therefore be undertaken as early as possible so as to reduce the tension whenever necessary.

Treatment. Therapy given immediately after inflammation recedes consists in prolonged instillation into the eye of dionine solutions, the application of yellow mercury ointment, repeated courses of tissue therapy (45-50 injections of aloe extract in daily 1 ml doses), and 15-20 sessions of electrophoresis with a 3-5 per cent potassium iodide solution or with aloe extract.

Persistent central opacities of the cornea with a transparent periphery may require *optic iridectomy*, i.e., excision of part of the iris to make an opening for the passage of light into the eye. The best effect is produced by *transplantation of the cornea*, an operation which has become widely popular through the efforts of Academician V. P. Filatov and his collaborators. This operation can also be performed in children, and the earlier the better, since it makes normal development of the visual functions possible.

The operation is essentially as follows: a special trephine is used to excise a disc-shaped portion of opaque scar-tissue from the wall-eye, and an identical flap of cornea taken from a cadaver's eye and preserved for two or three days at a temperature of 2-4°C is grafted onto the defect. The transplanted flap is fixed with a preliminarily cut-out flap of conjunctiva. Recently near-complete and complete grafting of the cornea has been used. The graft heals readily and in many cases retains its transparency.

DISEASES OF THE SCLERA

Diseases of the sclera may be due to an inflammation of the superficial layers of its anterior part—*episcleritis* or of the deeper layers—*scleritis*.

Episcleritis is manifested by redness and swelling in a limited portion of the sclera, usually at the limbus. *Scleritis* (Fig. 63) is

identified by a cyanotic colour of the inflamed region and injection of the deeper vessels.

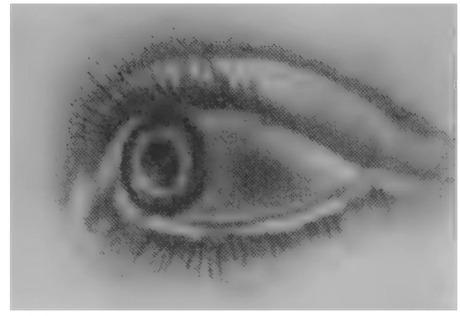

Fig. 63. Scleritis

Both diseases are accompanied by pain, moderate lacrimation and photophobia. The cornea is often involved, its deeper layers on the border with the limbus becoming hazy. Inflammatory symptoms are also noted in the iris. The diseases are associated with rheumatism, tuberculosis and gout, and less frequently with syphilis and brucellosis. Episcleritis usually has a favourable outcome, the inflammatory infiltration resolving without leaving a trace. Scleritis may, in the end, cause a thinning of the sclera, through which the vascular tunic is seen as a dark spot. Sometimes, an extension and bulging of the sclera is noted. Diseases of the sclera are liable to relapse and exacerbate frequently.

Treatment involves general measures applied taking account of the cause of the disease. Local therapy uses dionine, yellow mercury ointment, cortisone or adrenalin drops, or subconjunctival injection of 0.1 per cent adrenalin solution. Atropine is used for inflammation of the iris.

Therapy by UHF currents and heat is also beneficial.

11

DISEASES OF THE UVEAL TRACT

The extensive vascular network of the eyeball and the slow blood stream within it favour the retention of harmful agents circulating in the blood. Therefore, primary diseases of the uveal tract are endogenous in origin and are associated with tuberculosis, syphilis, influenza, brucellosis, malaria, relapsing fever, toxoplasmosis, rheumatism, and many other infections, as well as with metabolic disorders—diabetes and gout.

The aetiology of a disease cannot always be determined from its clinical picture, and therefore it is made clear by a detailed examination of the patient (analyses of blood, including Wassermann's test, urinalysis, x-ray examination, etc.).

In addition, there are exogenous diseases of the uveal tract concomitant with keratitis and penetrating wounds complicating their course.

IRITIS

Iritis, an inflammation of the iris, usually begins with a slight redness of the eyeball conjunctiva like that seen in conjunctivitis. Other typical signs of iritis, however, are different. These are sharp pain, lacrimation, photophobia, impairment of vision, contraction of the pupil, change in the pattern and shape of the iris due to the dilation of its vessels and deposition of exudate on its surface. A pale iris becomes green, and a dark iris rusty, which is especially noticeable when a sick and a healthy eye are compared. The floor of the anterior chamber is often covered by accumulated exudate, which causes dimness of the humor. Sometimes, the exudate becomes purulent (hypopyon) or sanguineous (hyphaema).

The above changes are characteristic of diffuse iritis which affects

the entire iris. In focal iritis (Fig. 64), nodular intumescences—tuberculae, papulae and gummae—are seen on the iris surface. The form and reaction of the pupil are changed: it is contracted, reacts weakly to light and dilates unreadily under the influence of mydriatics. Later, the presence of exudation causes adhesions between the iris and the crystalline lens (posterior synechiae) and the pupil becomes irregular in shape (Fig. 65). This is especially noticeable in dilation of the pupil after atropine instillation; it fails to dilate at all if the entire pupillary margin adheres to the crystalline lens (seclusio pupillae).

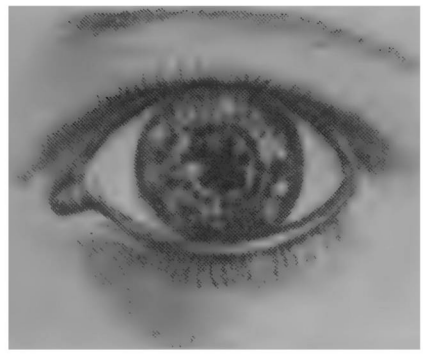

Fig. 64. Focal tuberculous iritis

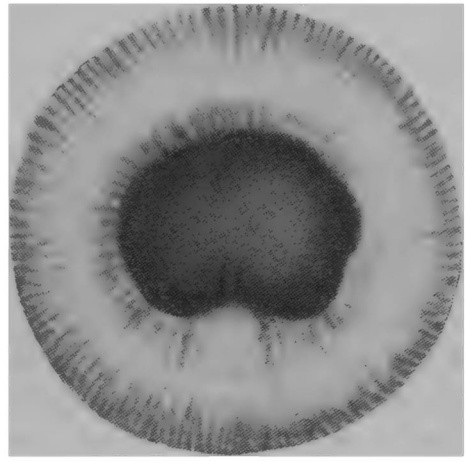

Fig. 65. Posterior synechiae in iritis

In the pupillary region, the exudate often forms a membrane of connective tissue (occlusio pupillae). This interferes with the flow of humor from the posterior to the anterior chamber. The humor accumulates behind the iris and causes bulging and secondary glaucoma threatening blindness, if attempts to reduce the intra-occular tension by therapy or surgery fail.

It should be emphasized that these complications can be prevented by adequate treatment given in due time. However, should they occur, the patient should be admitted to hospital immediately.

Upon comparison of all symptoms, diagnosis of iritis is usually not difficult. Errors, however, are likely: iritis is sometimes mistaken for acute conjunctivitis or, which is worse, for acute glaucoma, and the wrong therapy is prescribed. Iritis can be distinguished from conjunctivitis by the absence of exudation, the presence of a deep infiltration and, above all, by the characteristic changes in the iris and pupil (differential diagnosis between iritis and glaucoma is discussed in Chapter XIII).

IRIDOCYCLITIS

Iritis is very often joined by an inflammation of the ciliary body which is manifested by more intense pain and inflammatory symptoms, the appearance of precipitates (punctate collections of

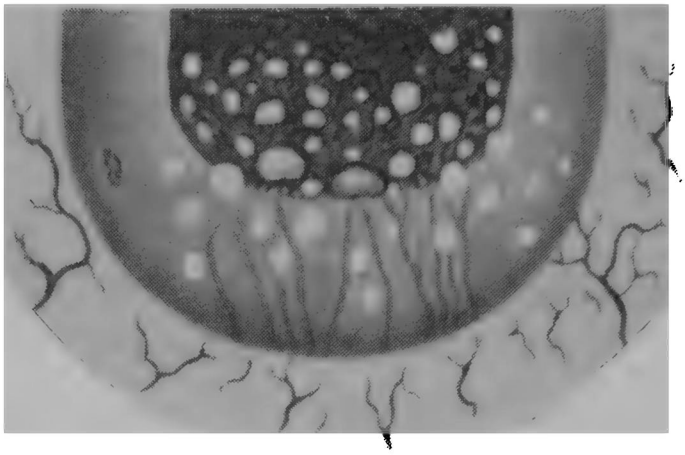

Fig. 66. Precipitates on the posterior surface of the cornea

exudate on the posterior surface of the cornea) (Fig. 66), haziness of the vitreous body, reduced intra-ocular tension and a certain loss of vision. A characteristic sharp pain is felt when pressure is applied to the eyes through the eyelid in the region of the ciliary body.

Adequate treatment may bring about complete resolution of opacities in the vitreous body. An unfavourable course of the disease is accompanied by accumulation of the exudate and formation of connective-tissue bands which wrinkle and are likely to cause detachment of the retina. A disorder in intra-ocular metabolism leads to opacity of the crystalline lens (complicated cataract), shrinkage or atrophy of the eye and blindness. Iridocyclitis is prone to frequent relapses often developing after chilling of the body.

Treatment. Before the cause of the disease is identified and specific therapy undertaken, the patient is treated with intramuscular injections of penicillin in novocain solution (in doses of 100,000 U three times a day) or streptomycin (in doses of 250,000 U twice a day) as well as with sulphonamides given per os. Derivative therapy is helpful: leeches on the temple, hot foot baths, saline purgatives. If there are no kidney diseases, osmotherapy is given in the form of intravenous infusions of a 10 per cent sodium chloride solution in a dose of 10 ml every other day (10-15 infusions per course of treatment). Local therapy is highly important. It consists above all in atropine instillations every one or two hours until the pupil is dilated and then at longer intervals to keep the pupil dilated. If atropine solution fails to give a sufficient effect, atropine ointment or even a grain of dry atropine can be applied. When applying dry atropine, the region of the lacrimal sac should be pressed with the finger to prevent the drug entering the lacrimal passages and thus passing to the nasopharynx, oesophagus and stomach; this prevents too early an absorption of atropine and poisoning. Atropine is very

effective in combination with other drugs, including adrenalin injected subconjunctivally in a 0.2 ml dose of 0.1 per cent solution. Today, successful use is made of subconjunctival injections of a cortisone suspension in a dose of 0.2 ml once every three days (4-5 injections in all) or of drop instillation of a 2.5 per cent cortisone solution three or four times a day.

The application of mydriatic drugs controls inflammation, prevents the formation of adhesions or causes their rupture, and averts secondary glaucoma. In this case, atropine should be replaced by pilocarpine, and if therapy is ineffectual surgery is required. It is very important to note any increase in the intra-ocular tension in due time, and therefore it should be checked during treatment.

CHOROIDITIS

This is an inflammation of the vascular coat, choroid, of the eye. At the onset, the disease gives no pain, and is manifested by a disorder of eyesight, with fluttering specks in the field of vision, distorted images of objects seen, and a certain loss of visual acuity. The ocular fundus is covered with greyish-white and pigmented foci located both in the centre and on the periphery, the retinal vessels passing above the foci.

Treatment. The choice of general therapy depends on the cause of the disease. Local therapy uses a dionine solution given in drops or subconjunctivally; in tuberculous conditions, streptomycin is administered subconjunctivally or by way of electrophoresis.

ENDOPHTHALMITIS

Endophthalmitis (purulent inflammation of the internal structures of the eye) occurs in general septic infections, purulent corneal ulcers complicated by infection, and penetrating wounds, resulting from extension of the disease to the internal structures of the eye. The patient complains of sharp pains in the eye and head and loss of vision. Examination reveals considerable oedema of the eyelids and the eyeball conjunctiva. This oedema forms a circular swelling round the cornea (*chemosis*). The cornea is lustreless and rough to the touch, with exudate or pus on the floor of the anterior chamber. The iris is dirty-green in colour, and the pupil shows a green tinge owing to the collection of pus in the vitreous body. The condition often extends to the external structures of the eye and orbital cellular tissue. The symptoms become worse, with bulging of the eyeball and disturbance of its motility (*panophthalmitis*).

Treatment. Intramuscular injections of antibiotics, sulpha drugs per os, and the application of heat. If no reversal of the condition is noted, *evisceration* (removal of the entire contents of the globe after cutting off the cornea) has to be undertaken to prevent the suppuration extending along the optic nerve and vessels into the brain.

To avert the spread of infection, antibiotic and sulpha drug therapy should be continued for at least five to seven days after surgery.

NEWGROWTHS OF THE UVEAL TRACT

These are commonly malignant pigmentary tumours which may be confined to any portion of the uveal tract. A tumour of the iris appears as a dark pigmentary mass, which grows in size and may extend beyond the limits of the eyeball and orbit. The tumour is liable to metastasize early to the visceral organs. At an early stage of the tumour, excision of the eyeball is required. In far-advanced cases it is recommended that the orbital contents be removed together with the eyeball (*exenteration of the orbit*); sometimes the paranasal sinuses are opened at the same time, and this is followed by deep x-ray therapy.

12

DISEASES OF THE CRYSTALLINE LENS AND THE VITREOUS BODY

For good vision the basic property required of the crystalline lens is transparency. When transparency is normal the pupil appears black; when the crystalline lens is opaque the entire pupil or its separate portions, depending on the degree of opacity, become grey. In transmitted light, partial opacities appear as dark spots or radiate bands against the red reflex in the pupil. If there is opacity of the entire crystalline lens, no red reflex in the pupil is noted.

DISEASES OF THE CRYSTALLINE LENS

Cataract

Opacity of the crystalline lens is called *cataract*. In the majority of cases, it involves disturbances of vision, the degree depending on the site and intensity of opacity. If there is complete opacity of the entire crystalline lens, vision is equivalent to the light sense, i.e., the patient is able to differentiate between light and dark only.

A cataract may be of a soft or hard consistency. The former is encountered in young persons who have no nucleus in the crystalline lens, and the latter in people above 30 when the nucleus is forming or has already formed. The choice of operation depends on the character of cataract.

Cataracts may be congenital or acquired.

Congenital cataracts, commonly partial, are not prone to progression. These are defects of intra-uterine development; they have different names according to the site of opacity. There are anterior or posterior polar cataracts in which the opacity is confined to the anterior or posterior pole of the lens; lamellar cataracts due to

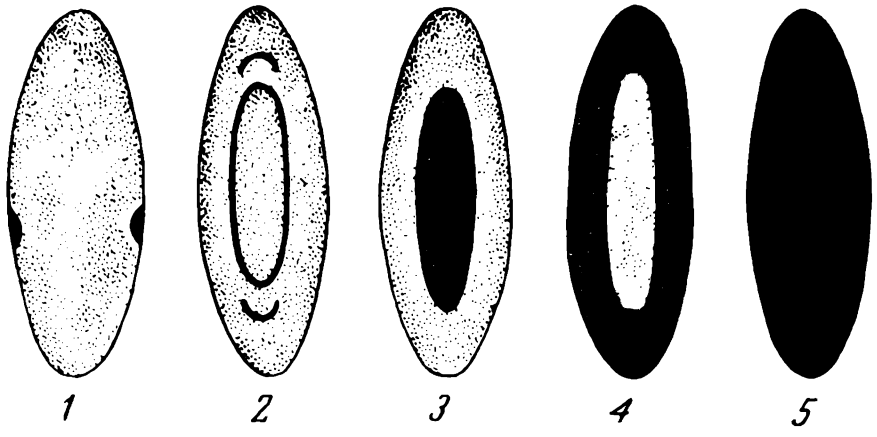

Fig. 67. Various types of congenital cataract
1—anterior and posterior polar cataracts; *2*—perinuclear lamellar cataract; *3*—nuclear cataract; *4*—cortical cataract; *5* complete cataract

alternate transparent and opaque layers of the crystalline lens; nuclear cataracts due to opacity of the centre (nucleus) of the crystalline lens; cortical cataracts due to opacity in the outer layers of the lens; complete soft cataracts in which there is opacity of the entire crystalline lens, etc. (Fig. 67).

Lamellar and especially *complete* cataracts are frequent causes of *congenital blindness.* The formation of these cataracts is favoured by intra-uterine diseases of the foetus. It has been found that toxoplasmosis, German measles, influenza, chicken-pox, mumps and some other virus infections sustained by women during pregnancy, particularly in the early months, may cause developmental defects in a foetus, including cataract.

Treatment is by means of early surgical interference. Therefore, a mother with a blind child should immediately be sent to a specialist.

Acquired cataracts run a progressive course and may result in complete opacity of the crystalline lens. The most frequent form among acquired cataracts is senile cataract, followed in order of frequency by cataracts due to diabetes, myotonia, tetany or associated with diseases of other parts of the eye.

Senile cataract is encountered in old people and runs a course consisting of four stages.

Primary cataract (cataracta incipiens) is characterized by partial opacity of the outer layers of the crystalline lens, which in transmitted light, against the red reflex in the pupil, look like spokes almost reaching the lens centre (Fig. 68). The loss of vision is insignificant. But in some cases, the opacity begins in the lens centre, causing a sharp loss of visual acuity. Patients with primary cataract complain of dark spots in the visual field and impaired vision.

In *intumescent* or *immature* cataract (cataracta intumescens) the opacities spread as far as the centre of the pupil and coalesce with one another. Examination in transmitted light reveals in places only the red reflex of the ocular fundus. The acuity of vision is markedly impaired. The transparency of the superficial layers of the crystalline lens is retained, and therefore a shadow of the iris is seen at the pupillary margin in lateral or focal illumination (Fig. 69).

In *a mature cataract* (cataracta matura), the opacity covers the entire crystalline lens, the pupil appears grey (Fig. 70*a* and *b*), the iris shadow is absent, and vision decreases to the degree of the light sense with correct projection.

In *an overripe cataract* (cataracta hypermatura) the outer layers of the crystalline lens dissolve, and the nucleus wrinkles and descends to the capsule floor.

It is very important to know about the maturity of a cataract to make a wise decision on surgery, which is the principal method of treatment.

Treatment. Once it has become ripe, a cataract can be extracted. If an immature cataract is removed only the nucleus is extracted. The remaining fibres of the crystalline lens later become swollen and hazy and, together with the remnants of the capsule, form a secondary cataract—a membrane in the pupillary region requiring further surgical interference. Extraction of an overripe cataract also involves difficulties. That is why a patient with cataract should be kept under observation by an ophthalmologist so that surgery is carried out at the right time.

Prior to cataract extraction, as in any other surgical interference involving excision of the eyeball, the patient should be subjected to a thorough examination.

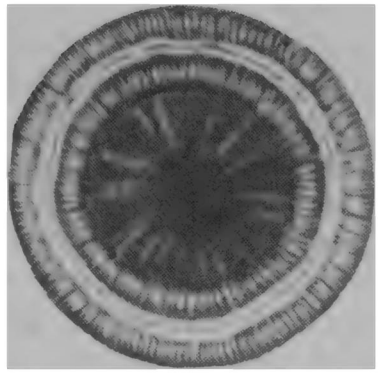

Fig. 68. Primary cataract in transmitted light

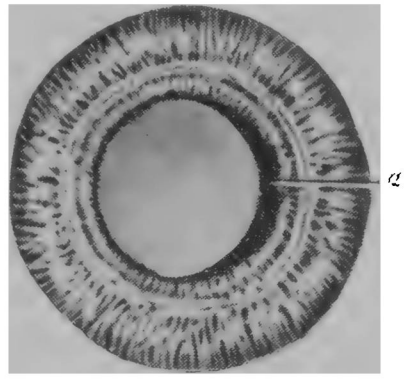

Fig. 69. Immature cataract in focal light
a—shadow of the iris

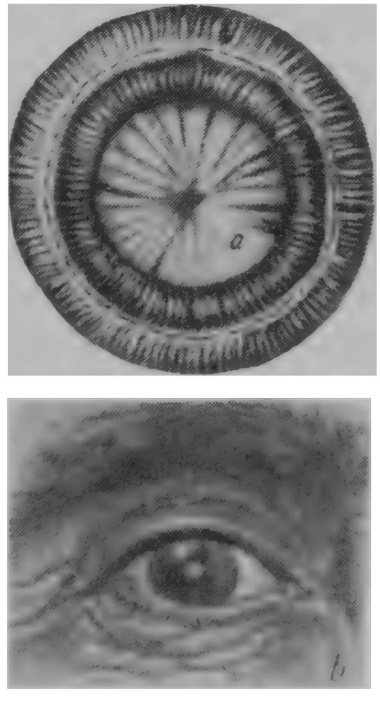

Fig. 70. Mature cataract
a—entire lens is opaque;
b—pupil is grey

Extraction of the crystalline lens is performed by an extracapsular method, i.e., after its capsule has been opened, or by an intracapsular method, i.e., by removal of the crystalline lens together with its capsule. The latter method is technically more complicated, but it ensures a better outcome with regard to subsequent functional activity and enables an immature cataract to be extracted.

The operation leaves a delicate scar on the site of the excised cornea, the anterior chamber becoming deeper, and the iris trembling with movement of the eyeball because of the absence of support. A defect (coloboma) is visible in the upper half of the iris. The pupil is black. The condition of the eye following extraction of the crystalline lens is called *aphakia*.

After an operation the patient is prescribed spectacles with 10.0-12.0 D convex lenses for permanent wear. With such correction, the acuity of vision may reach a value of several decimals or even 1.0. When the crystalline lens is removed the eye loses its power of accommodation; spectacles prescribed for reading and close work have lenses which are stronger by 3.0-4.0 D than those for distances. In cases of hypermetropia, stronger lenses are prescribed for wear before an operation; in cases of myopia, weaker lenses corresponding to the degree of refraction anomaly. Spectacles are not prescribed after an operation on one eye, if vision in the other eye is good enough, because the patient will find it troublesome to wear a strong lens sharply magnifying the image in one of his eyes.

This, however, does not mean that surgery for a unilateral cataract should be avoided, since after an operation the eye will have slight vision even without a lens and can help to enlarge the general visual field; if vision in the other eye is lost it will become the only means of seeing. This is very important to the patient, since a senile cataract is usually a bilateral process, though not necessarily developing at the same time in both eyes. Today contact lenses applied directly to the eyeball have begun to be used after the extraction of a cataract in one eye.

Secondary successive or membranous cataracts appear after an operation due to inadequately careful removal of the lens-matter or its capsule, after resolution of a traumatic cataract, and as a sequel to postoperative iridocyclitis when the pupil and the region of the newly-formed defect (coloboma) are covered by an exudative membrane. This results in a partial loss of vision, and sometimes in an increased intra-ocular tension.

Treatment involves surgical removal of the membrane.

Traumatic cataracts appear as a result of a discontinuity of tissue due to a penetrating wound of the eye or contusion. In young people opacity of the lens-matter sets in early, and is accompanied by a loss of vision, even to the extent of losing the light sense.

In certain cases, particularly in children, the opacity of the lens-

matter may resolve. If this fails to occur, and especially when there is increased intra-ocular tension or a sharp irritation of the iris, an operation is required to allow the lens-matter to escape.

Complicated cataracts follow diseases of other parts of the eye (glaucoma, uveitis, pigmentary degeneration of the retina, detachment of the retina, high-degree complicated myopia, etc.), and are also often associated with systemic diseases (diabetes, tetany, sequels of irradiation, etc.). This type of cataract is extracted by surgery which is preceded by therapy corresponding to the aetiology of the disease.

Dislocation of the crystalline lens may be congenital or traumatic. Examination reveals a deep anterior chamber and trembling of the iris, while the black margin of the displaced lens is visible during examination in transmitted light. Congenital dislocation of the crystalline lens usually occurs in both eyes.

Treatment for severe loss of vision or secondary glaucoma is surgical.

DISEASES OF THE VITREOUS BODY

Normally rays of light reach the retina freely through the transparent vitreous body. In inflammation of the uveal tract, intraocular haemorrhages, traumas of the eye and infection of its internal structures, the vitreous body is found to contain opacities looking like flakes or threads and moving even when the eye is still. These opacities are traces of haemorrhages and exudations or result from shrinkage of the fibrils of the vitreous body.

Treatment is chosen according to the principal cause of the disease. Resolution of opacities is favoured by dionine solutions, electrophoresis with potassium iodide, intravenous infusions of a 10 per cent sodium chloride solution in 10 ml doses every other day (10-15 infusions in all), tissue therapy by grafting preserved tissue, or 45 subcutaneous injections of aloe extract in daily 1 ml doses, as well as by blood transfusion. These methods of therapy are effectively combined with repeated injections of oxygen under the eyeball conjunctiva (every other day, 15-20 injections per course of treatment).

13

GLAUCOMA

One of the most frequent causes of incurable blindness is glaucoma, which accounts for about 4 per cent of the total incidence of eye diseases. Glaucoma is mainly encountered in elderly persons, but is sometimes found in juveniles and children also.

The term "glaucoma" is derived from the Greek "glaucos"—green; the disease owes its name to the yellow-green reflex seen in the pupil during an acute attack.

The principal symptom of glaucoma, determining all its other manifestations, is a heightened intra-ocular tension proceeding to a gradual lessening of visual power, restriction of the field of vision, impairment of adaptation and atrophy of the optic nerve.

The intra-ocular tension is related to the pressure from the ocular contents and the blood pressure in the capillaries. The normal intra-ocular tension fluctuates within a range of 18 to 28 mm Hg; in the morning it is usually higher, and in the evening lower, the difference being 3-5 mm. A rise in intra-ocular tension as well as an increase in the difference between its morning and evening values is evidence of a disturbance in its regulation, characteristic of glaucoma.

Glaucoma may come on suddenly, without any apparent cause, in a hitherto normal eye (primary glaucoma) or may be consequent upon some other ocular disease (secondary glaucoma)—changes interfering with aqueous drainage from the eyeball, for example, adhesion of the iris to the cornea or the crystalline lens, etc.

The most important factor in the pathogenesis of glaucoma is a metabolic disturbance in the intra-ocular fluid which is believed to be a product of ultrafiltration from the vessels of the retina and the ciliary body. The basic role in this process is played by the capillary endothelium. In the late 19th century it was generally

agreed that glaucoma is "a diseased eye in a diseased body". Recently convincing evidence has been obtained on the relationship between the regulation of intra-ocular tension and the condition of the central nervous system and the neurovascular reflex in the eye.

The origin of glaucoma and aggravation of its course are favoured by various adverse factors: diseases of the cardiovascular, nervous and endocrine systems, infections and intoxications, hard manual work, great mental strain, negative emotions, etc.

THE CLINICAL PICTURE AND CLASSIFICATION OF GLAUCOMA

Primary glaucoma may have different manifestations. In some cases there are signs of disturbance of the blood circulation in the eye with marked symptoms of stasis in the vessels. This form is known as *congestive*, and patients complain of progressive loss of vision, a misty visual image, difficulty in orientation in space, particularly in darkness, and pains in the head and the eye. A characteristic complaint is of a coloured halo seen round lights. The earliest, but not specific, complaints are those of lacrimation without cause, early visual fatigue in reading and the resulting need for a frequent change of spectacles.

Examination of the patient reveals expansion of the venous network around the cornea, a decrease in the depth of the anterior chamber, dilation of the pupil, atrophy of the iris, and a change in its colour and relief. As the disease progresses atrophy and excavation ("cupping") of the optic disc develop (Fig. 71*a*, *b*). The acuity of vision decreases and in refraction anomalies is inadequately corrected with lenses. The visual field is at first narrowed on the nasal side and then gradually narrows concentrically; adaptation is impaired.

Tonometry undertaken twice a day shows an increase in pressure, sometimes up to 40-50 mm Hg, and a wide range in fluctuation between the morning and evening indices (more than 10-15 mm).

In another, simple (non-conjestive) form of glaucoma the patient complains only of a gradual loss of vision. The anterior portion of the eye may show no changes, and the characteristic changes in the optic disc are found only on the ocular fundus. Early diagnosis of simple glaucoma, necessary if the patient is to be given effective help, can be established only by means of examination of the intra-ocular tension and the visual field.

According to the conventional classification (B. L. Polyak), glaucoma is known to have several *stages* and different forms. Initial glaucoma is characterized only by the above-described symptoms, which are mild and unstable. For example, misty vision and a head-

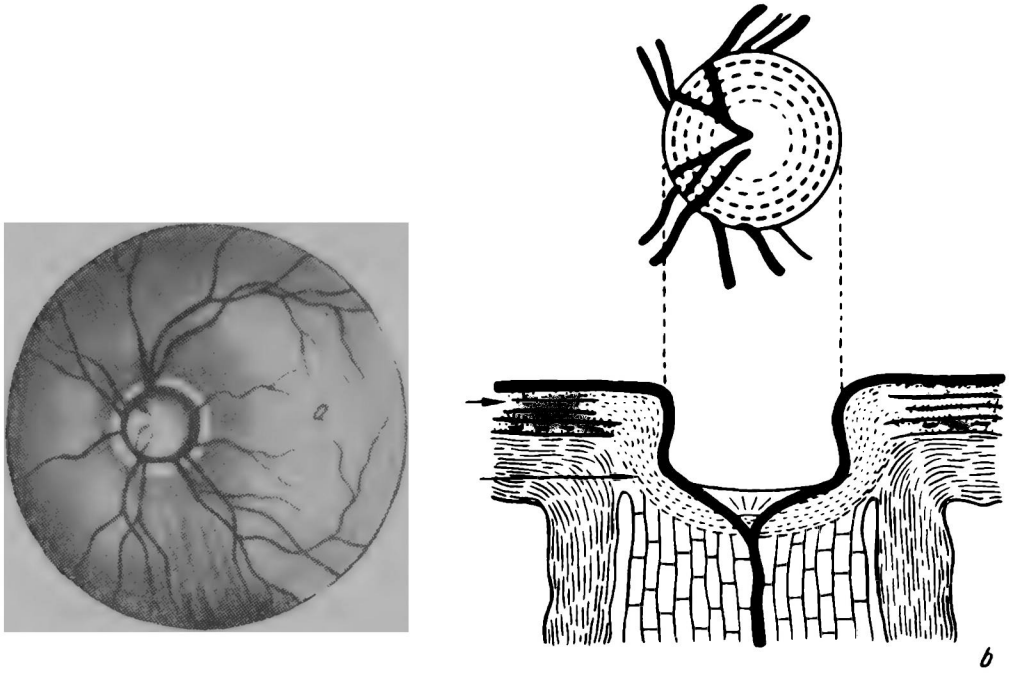

Fig. 71. Glaucomatous excavation of the optic disc
a—ophthalmoscopic picture; *b*—section through the optic disc

ache may come on suddenly and pass off shortly. The changes described in the anterior portion of the eye can be found only during such a mild attack, after which they disappear. The acuity of vision and the visual field as well as the ocular fundus may remain unchanged; however, periodic measurements of the intra-ocular tension can reveal an increase and identify glaucoma.

Advanced glaucoma is characterized by stable changes in the anterior portion of the eye and excavation of the optic disc. There is no decrease in the intra-ocular tension, the acuity of vision is impaired, and the field of vision on the nasal side shows a stable narrowing.

In *far-advanced glaucoma* the loss of vision is aggravated further, the visual field is markedly narrowed on all sides, and atrophy of the optic nerve appears.

The above changes are caused by the death of the fibres of the optic nerve as a result of increased intra-ocular tension and trophic disorders. Then follows a loss of vision to the degree of simple light perception—*almost absolute glaucoma*—and, finally, *absolute glaucoma* sets in with total loss of photoreception, and blindness.

The classification also lists a state of *compensation* of glaucoma under the influence of therapy. There is *compensated glaucoma* with normal intra-ocular tension, *sub-compensated glaucoma* with characteristic fluctuations of the intra-ocular tension within a range of 29 to 35 mm Hg, *noncompensated glaucoma* in which the intra-ocu-

lar tension is greater than 35 mm Hg, and *decompensated glaucoma*, or an acute attack with severe signs of glaucoma and a very high intra-ocular tension (50 mm Hg and higher).

The above stages of the disease develop if compensation of the intra-ocular tension is absent. This commonly occurs when the patient applies late for medical aid and when treatment is inadequate or irregular.

ACUTE ATTACK OF GLAUCOMA

This attack may come on suddenly, often after nervous shock, chilling of the body, physical overstrain, various diseases, etc. An acute attack is less frequently the initial sign of glaucoma, and commonly develops when the disease has been in progress for some time.

During an attack the patient has an excruciating pain in the eye and the dependent half of the head, referred to the teeth. This is often followed by vomiting and sometimes by fever. These symptoms may often be the cause of a diagnostic error as a result of which patients with an acute attack of glaucoma are placed in a therapeutic or neurological hospital. During an attack, considerable palpebral oedema and lacrimation are noted. The eyeball conjunctiva is markedly hyperaemic due to stasis in the anterior ciliary veins, forming a corolla about the lustreless, oedematous and rough cornea, whose sensation is sharply diminished or lost altogether. The anterior chamber is very shallow or absent altogether. The pupil shows a yellowish-green reflex, is dilated and reacts weakly to light, if at all. The ocular fundus does not permit ophthalmoscopy because of severe oedema of the cornea and the vitreous body. The intra-ocular tension is markedly heightened, and the eyeball is as "hard as a stone". There is a large loss of vision, sometimes extending to the light sense. This clinical picture is characteristic of an acute attack of glaucoma; lack of care, however, may entail diagnostic errors with fatal consequences for the visual power in the sick eye. It is especially dangerous to mistake glaucoma for iritis, since the administration of atropine will increase the intra-ocular tension which is already greatly raised. It should be emphasized again that *at the slightest suspicion of glaucoma, atropine should by no means be instilled into the eye.*

The following table for a differential diagnosis between glaucoma and iritis has been suggested by Academician M. I. Averbach.*

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* The text in the table is somewhat altered without detriment to its content.—*Ed.*

Acute Attack of Glaucoma	Acute Iritis
Intra-ocular tension raised	Intra-ocular tension normal or slightly lowered. May sometimes be slightly increased
Cornea invested with diffuse haziness, to the degree of complete opacity	Cornea without marked changes
Its surface covered by dots	
Deep injection unmarked	Considerable injection of the corneal limbus vessels
Anterior ciliary veins prevail	
Pupil is wider than in the fellow eye	Pupil is narrower than in the fellow eye (if not dilated with atropine)
Corneal sensation diminished	Corneal sensation normal
Anterior chamber shallow	Anterior chamber of normal depth
Radiating pains prevail in the forehead, jaws, nape	Pains mostly confined to the eye region
Complaints of coloured halo and dim vision	No complaints of coloured halo. Impairment of vision possible

The outcome of an acute attack of glaucoma depends on prompt and adequate therapy and on the stage of the disease. In the initial stage of glaucoma, all symptoms may disappear and the vision return to normal; cases are known, however, of incurable total blindness caused by the first attack of the disease. Following an acute attack, the intra-ocular tension usually remains elevated, and the disease takes a chronic course, sometimes with intermittent acute attacks. Errors in the diagnosis of simple glaucoma are even more frequent because the sick eye is normal in appearance.

The advanced age of a patient, the absence of complaints other than those of impaired vision, and the greyish reflex in the pupil sometimes mislead the examiner, who mistakes glaucoma for cataract and advises the patient to wait patiently until it becomes ripe and operable. Such advice is disastrous, since blindness from glaucoma thwarts all efforts at therapy. With due attention, however, symp-

toms of the disease are discoverable in simple glaucoma also. They are seen as a slightly shallow anterior chamber, a somewhat widened and greyish pupil uncharacteristic of the patient's age (in old persons the pupil is usually narrower than in young ones), and "cupping" of the optic disc. Especially typical signs are an increased intra-ocular tension and a changed field of vision, which are not characteristic of cataract. Besides, cataract can be differentiated from glaucoma by examination of the eye in transmitted light since cataract will show no red reflex in the pupil.

As has been mentioned above, blindness due to glaucoma results from progressive atrophy of the optic nerve caused by a raised intra-ocular tension and the inevitable associated trophic disturbances. The blind eye may cause no painful sensations for a long time but disturbance of corneal nutrition often leads to detachment of the corneal epithelium with the formation of blebs (keratitis bullosa).

These blebs break down leaving raw areas subject to ulceration and likely to cause perforation of the cornea and prolapse of the inner coats of the eye.

DIAGNOSIS, PROPHYLAXIS AND TREATMENT OF GLAUCOMA

The diagnosis of glaucoma is based on the above-mentioned clinical signs of disturbance of the visual functions, such as deterioration of visual acuity, defects in the visual field and especially a heightened intra-ocular tension. Tonometry and the plotting of the elastonometric curve are performed in an ambulant centre. If a suspicion of glaucoma based on clinical symptoms is not confirmed by clear tonometric findings, the patient should be hospitalized. Patients with apparent glaucoma should be placed in a hospital for study of the progress of the disease, selection of the method of therapy, estimation of the effectiveness of therapeutic measures and, whenever required, for surgery. The basic procedure in an examination of a glaucomatous patient is measurement of the intra-ocular tension, which is performed twice daily (at 7 a.m. and at 7 p.m.), and in certain cases even more often. The tension indices expressed in mm Hg are plotted in the form of a curve analogous to that showing fluctuations in fever. In addition, other visual functions are also examined. If the diagnosis is uncertain, various tests are used obstructing (loading tests) or facilitating (unloading tests) intra-ocular fluid drainage.

An important feature in the *prevention* of the onset and progression of glaucoma is early detection of the disease. The intra-ocular tension should therefore be examined in all persons above 40 ap-

plying for medical aid for whatever reason. Should it prove to be above 25 mm Hg, particularly in a patient complaining of headache, deterioration of vision, and a coloured halo seen around lights, the attention of an ophthalmologist is required immediately. In addition, active detection of glaucoma cases is carried out during prophylactic examinations and tonometry is conducted by physicians and ancillary medical personnel among all persons above 40 under the care of a particular medical centre or at institutions, etc. This helps to detect glaucoma in 1-2 per cent of persons examined.

Patients with glaucoma are registered at the district medical centre and with the district ophthalmologist. They are given regular therapy and a check-up (at least once monthly) on the condition of the intra-ocular tension and the visual functions. The medical assistant or nurse sees that the patient fulfils the doctor's instructions concerning therapy, working and everyday routine and that he consults the ophthalmologist regularly.

For active control of glaucoma the population should be informed about this disease; the medical service uses various means of health education for this purpose (discussions, lectures, press, radio, etc.).

As a result of extension of dispensary services and active detection of glaucoma cases there has been a marked increase in the number of applicants for medical aid with early signs of the disease, and in the proportion of patients with stable compensation of the process following regular therapy. The outlook of the disease has become more favourable and the rate of blindness due to glaucoma has decreased.

Congenital glaucoma (hydrophthalmus congenitus) develops in children with a congenitally underdeveloped or atresic Schlemm's canal. Symptoms of the disease can be seen at birth or in the early months of life as a marked enlargement of the cornea under the influence of an elevated intra-ocular tension and thinning of the sclera, through which the vascular tunic (choroid) is visible (Fig. 72). The cornea is hazy due to oedema and trophic disturbances. The anterior chamber is very deep and the iris is atrophied. Such children should be sent immediately to an ophthalmologist for an operation, since otherwise blindness may soon follow as a result of optic nerve atrophy.

The principal aim in the treatment of glaucoma is reduction and regulation of the intra-ocular tension. Drugs constricting the pupil and decreasing the intra-ocular tension (*miotics*), such as pilocarpine, eserine, furamon (farfural trimethyl ammonium), phosphacol (diethyl p-nitrophenyl phosphate), etc., are used for this purpose. The most common drug is pilocarpine which, should it prove inadequate, is combined with other drugs to intensify each

other's effect. The frequency of instillation of miotics depends on the form and stage of the disease and especially on the degree of compensation. For example, in an acute attack of glaucoma, pilocarpine is instilled into the eye every 15 to 30 minutes and, when the acute symptoms abate, once every one or two hours and less often; at the same time eserine or phosphacol is instilled two or three times a day.

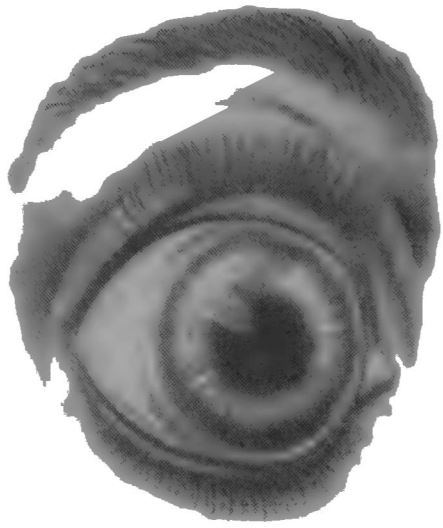

Fig. 72. Congenital glaucoma

In other cases miotics are instilled three or four times a day, depending on the value of the intra-ocular tension. The routine of treatment and type of drug are best chosen during examination of the patient in a hospital when a constant check is made on the diurnal fluctuations in the intra-ocular tension.

During an acute attack of glaucoma *derivative therapy* may also be applied by means of hot foot baths, saline purgatives (solution of 25 g of effervescent magnesium sulphate or sodium sulphate) and leeches on the temple. If in an acute attack of glaucoma attempts to reduce the intra-ocular tension fail for as long as one day, surgery is required. If the tension does not return to normal under the influence of therapeutic remedies, an operation is undertaken for other forms of glaucoma also. Surgery for acute glaucoma consists in removing part of the iris (iridectomy), which serves to free the angle of the anterior chamber and improves humor drainage from the eye. In other forms of glaucoma (except absolute) a fistula is formed in the sclera (fistulizing operation) to provide a new route of humor drainage under the conjunctiva. Other operations are also known.

In addition to the above treatment, regular courses of tissue therapy are also helpful for glaucoma, particularly in the pre- and postoperative periods. This therapy consists in intravenous or intramuscular injections of a 5 per cent thiamine (vitamin B_1) solution in daily 1 ml doses or of sodium chloride solution once every two days. Besides, patients with glaucoma often need appropriate treatment of the cardiovascular, nervous and endocrine systems and of the gastrointestinal tract.

A particular regimen is also an important part of glaucoma treatment. The patient should avoid excitement, nervous shocks, overheating or chilling of the body, staying for a long time near a hot kitchen-range, working in a stooping posture, and physical and men-

tal overexertion. It is important to evacuate the bowels regularly. The patient is recommended to take a milk and vegetable diet with a limited quantity of water (4-5 glassfuls per day). Strong drinks and smoking are strictly forbidden. Light physical exercise is desirable. In secondary glaucoma, therapeutic or surgical treatment is directed towards removing the principal cause of an increased intra-ocular tension.

Prompt diagnosis of secondary glaucoma and prescription of vigorous treatment, including surgery, are highly important because such patients often lose their eyesight from elevated intra-ocular tension occurring as a complication rather than from the principal disease (corneal ulcers and wall-eye, iridocyclitis, penetrating wounds, etc.).

14

DISEASES OF THE RETINA AND OPTIC NERVE

DISEASES OF THE RETINA

Diseases of the retina result in a sudden or gradual lessening of visual power. When there are lesions of the central part of the retina—the macula lutea and its surrounding tissues (paramacular) the size and shape of objects are distorted, the colour-sense is disturbed, there is a considerable loss of vision at a distance, while close reading becomes impossible. Changes of the retina are detected by ophthalmoscopy.

These changes involve the retinal vessels or tissue. In the latter case, the retina shows diffuse or focal opacities, haemorrhages and pigmentation. These develop in cases of vascular hypertension, kidney and blood diseases, tuberculosis, syphilis, brucellosis, diabetes, toxicoses of pregnancy, and many other pathological conditions. Discovery of retinal changes enables the ophthalmologist to take an active part in diagnosing the above diseases.

Circulatory Disorders in the Retina

Special attention should be given to retinal diseases associated with circulatory disorders due to *acute obstruction of blood vessels*, which may occur as a result of embolism, spasm, and arterial or venous thrombosis.

Obstruction of the central retinal artery results in a sudden total loss of vision. Ophthalmoscopy (Fig. 73) reveals marked constriction of the arteries and anaemia of the ocular fundus, particularly in the region of the macula lutea, against whose pale background a characteristic cherry-red spot is visible. If an embole obstructs one of the branches rather than the trunk of the central retinal artery, the signs of anaemia and vascular stricture are seen in a limited area of the ocular fundus along the course of the vessel, vision being

only partially lost and the visual field being partly blotted out. Embolism develops with heart defects as a result of obstruction of a retinal vessel by an embole breaking off from the heart valve. A similar picture, often with haemorrhagic complications, is seen in *thrombosis of the artery* resulting from endarteritis associated with atherosclerosis.

A spasm of the central retinal artery or one of its branches may develop in a young person as a manifestation of a functional disease of the central nervous system. The clinical picture is like that of embolism but, unlike the latter, organic heart disease is absent and the loss of vision is transient.

Thrombosis of the central retinal vein is marked by sudden sharp deterioration of vision, and is encountered mainly in old persons suffering from atherosclerosis and vascular hypertension. The veins of the ocular fundus are widely dilated, and haemorrhagic spots are seen along their course.

Treatment. Remedial measures are prescribed for the principal disease by a therapeutist or a neuropathologist, and the patient is kept under observation by the ophthalmologist. Local therapy for venous thrombosis uses pilocarpine because of the likely danger of an increase in the intra-ocular tension. Embolism is treated with retrobulbar injections of a 0.1 per cent atropine solution in doses of 0.5-1 ml, with a total of 5 to 10 injections per course of treatment. Nicotinic acid is given per os or intravenously and anticoagulants are given per os to induce dilation of the vessels. Treatment should be begun as early as possible; the prognosis in these diseases, however, is nearly always unfavourable.

Pathological Changes of the Retina and Retinal Vessels in Certain Systemic Diseases

Changes of the retina and the retinal vessels in *vascular hypertension* depend on the patient's general condition. In the initial stage of the disease the retinal vessels are narrowed or dilated and the minutest veins are sinuous. In more advanced vascular hypertension other signs are unevenness of the vessel walls and at places emptiness of vessels. As a result of sclerosis, veins sink into the retinal tissue to some extent at the intersections of arteries with veins (Salus' symptoms of I, II and III degree). In severe vascular hypertension, the retina shows haemorrhages, a degenerated macula lutea, and white spots of variable shape. A similar picture can be seen in nephritis attended by an increase in blood pressure. Symptoms of vascular hypertension in the eyes are important for the diagnosis and prognosis of the principal disease; therefore, all patients with vascular hypertension should be sent to an ophthalmologist for examination of the ocular fundus.

Fig. 73. Embolism of the central retinal artery

Fig. 74. Pigmentary degeneration of the retina

Retinopathy of pregnancy, which is associated with intoxication and hypertension, is commonly found in the later half of pregnancy. Termination of pregnancy may be followed by complete recovery. The changes in the ocular fundus are analogous to those described above. Attention should be given to pregnant women's complaints of impaired vision and they should be sent without fail to an ophthalmologist, who in certain cases has to consider abortion so as to save the patient's life and preserve her eyesight.

Characteristic changes of the retina are seen in severe forms of **diabetes, leukaemia, pernicious anaemia, etc.**

A very severe disease is *periphlebitis*, of the retinal vessels, an inflammation of the walls of veins. Vision is gradually lost as a result of repeated vitreous haemorrhages which resolve slowly and finally produce adhesions in the vitreous body with subsequent **detachment of the retina.**

Pigmentary Degeneration of the Retina

Pigmentary degeneration is one of the most severe diseases of the retina. Its aetiology is obscure. It usually begins at a young age and slowly progresses to total blindness. The first sign of the disease is impaired vision in dim light and a narrowed field of vision. Later deterioration of central vision follows. Multiple pigmentary foci resembling bony corpuscles are seen on the periphery of the ocular fundus. The retinal vessels are markedly narrowed, and the optic disc is pallid (Fig. 74). In view of the gradual and steady progression of the disease, its outlook is very serious.

Treatment. Regular courses of tissue therapy can delay the onset of blindness. Cod-liver oil, riboflavin, ascorbic acid, and vitamins A and E are given per os, and thiamine is injected intravenously or intramuscularly. Repeated courses of retrobulbar injections of a 0.1 per cent atropine solution and subcutaneous injections of a 0.1 per cent strychnine solution are also given.

It should be remembered that nyctalopia may have a transient character in persons with vitamin A deficiency. A characteristic sign distinguishing this form from nyctalopia due to pigmentary degeneration of the retina is its sudden rather than gradual onset. At the same time, the visual field and the ocular fundus remain unchanged. When cod-liver oil, vitamins A and E and food rich in them are taken, recovery is quick.

Detachment of the Retina

Detachment of the retina (ablatio retinae) sets in with a sudden sharp loss of vision to the extent of total blindness. At first the patient notes a shadow appearing in the visual field of one eye when gazing in a fixed direction. The most common cause of detachment

of the retina is its rupture or tearing off at the site of attachment causing the vitreous fluid to pass into the space between the retina and the choroid. Rupture of the retina occurs with trauma of the eye, degenerative changes of the retina in old age, in persons with progressive myopia, etc. A very frequent cause of retinal rupture in myopia is trauma or physical overstrain. That is why persons with high-degree myopia should for prophylactic purposes be relieved from hard manual jobs and encouraged to refrain from heavy physical exercise.

Retinal detachment is identified by the appearance against a red reflex in the pupil of a V-shaped grey flap of vascilating detached retina on which dark tortuous vessels are visible. The intra-ocular tension is often decreased.

Treatment of retinal detachment involves surgery, an early operation being the most effective. Patients with suspected retinal detachment should therefore be sent immediately to an ophthalmologist.

The operation consists essentially in draining the subretinal fluid and providing conditions for an adhesive inflammation of the retina with the underlying tissues. For this purpose the sclera is cauterized by surgical diathermy and then punctured at the site of the retinal detachment and rupture. Other operations have also been used during the past few years.

After the operation a binocular bandage is applied. The patient must lie in bed on his back for as long as 14 to 21 days. Following an operation, vision may return to normal or improve considerably. In view of the likelihood of a relapse the patient should subsequently avoid physical exertion and be kept under observation by an ophthalmologist.

Newgrowths of the Retina

Retinoblastoma belongs to the group of primary malignant tumours of the retina, usually congenital, which may sometimes affect both eyes. The disease is commonly encountered in children under 5 years of age. It is usually noticed by the child's parents from a green reflex in the dilated pupil. There is total loss of vision owing to retinal detachment and the intra-ocular tension is raised by the tumour. Retinoblastoma is liable to rapid growth and metastases ending in death. The patient can only be saved by early diagnosis followed by removal of the eye or the orbital contents and subsequent radiation therapy.

DISEASES OF THE OPTIC NERVE

These diseases may be inflammatory (*neuritis*), congestive (*choked disc*) and degenerative (*atrophy* of the optic nerve).

The optic nerve may become involved in meningitis, encephalitis, arachnoiditis, brain tumours, multiple sclerosis, rheumatism, influenza, malaria, syphilis, tuberculosis and other infectious diseases, in intoxication with methylated alcohol, plasmocide (pamaquine naphthoate), lead, etc., in inflammations of the paranasal sinuses, diseases of the eyeball (choroiditis, retinitis), and the orbit (inflammations and tumours).

Identification of the disease and its aetiology requires a comprehensive examination of the patient, particularly the visual functions. Sometimes changes in the optic nerve are early signs of a disease of the central nervous system.

Inflammation of the optic nerve (neuritis nervi optici) comes on with a sudden deterioration of vision, contraction of the visual field and dark spots (scotomata) seen in the latter. The optic disc is hyperaemic, its outlines are blurred, the retina around the optic disc is oedematous, the retinal veins are dilated, and haemorrhages are often seen along the course of the veins (Fig. 75, see insert on p. 151).

Treatment depends on the aetiology of the disease. In addition, intramuscular antibiotic injections, intravenous infusions of a 40 per cent glucose solution with a 40 per cent urotropin solution and a 5 per cent thiamine solution are applied, as well as blood transfusions, tissue therapy and leeches on the temple.

Choked disc is the major symptom of a rise in intra-cranial pressure likely to be caused by a brain tumour, a gumma, a hydatid cyst, a cyst, or inflammatory processes in the brain tissue and meninges. At the outset choked disc does not produce a visual disorder, and is found only in examination of the ocular fundus. Later the visual power lessens and the field of vision narrows. Ophthalmoscopy reveals an enlargement and mushroom-shaped protrusion of the optic disc (Fig. 76, see insert on p. 151). The dilated and tortuous veins seem to be "climbing" the optic disc, and haemorrhages are seen along their course.

Treatment is chosen according to the cause of the disease, and is administered by specialists, commonly neuropathologists or neurosurgeons.

Atrophy of the optic nerve may be primary (in tabes, brain syphilis, multiple sclerosis, progressive paralysis, trauma of the optic nerve itself, pigmentary degeneration of the retina, methyl alcohol poisoning, etc.) or secondary to an inflammation of the optic nerve or choked disc. In this condition, the disc appears grey or white (Fig. 77, see insert on p. 151), and the retinal vessels are narrowed. Whatever the cause of optic nerve atrophy, there is a severe loss of central and peripheral vision.

Treatment depends on the aetiology of the disease. It is important to remember that in the case of syphilitics with atrophy of the

optic disc, mercury drugs and novarsenol (neoarsphenamine) should be applied with great caution as they can have a harmful influence on the residual functions of the optic nerve. Therefore, in the treatment of syphilis, especially neurosyphilis, it is necessary to watch the condition of the ocular fundus and the visual functions.

The vital activity of the optic nerve is favoured by regular repeated courses of tissue therapy, subcutaneous injections of a 0.1 per cent strychnine solution in increasing doses (from 0.2 to 1 ml), intravenous infusions of glucose solution with thiamine and nicotinic acid, intramuscular injections of vitamins B_1, B_6, B_{12}, and novocain. Riboflavin is given per os.

The outlook of optic nerve atrophy is grim because the disease constantly progresses, central vision deteriorates as far as total blindness, and the visual field narrows. The patient should be kept under permanent observation by an ophthalmologist and given regular repeated treatment.

15

DISEASES OF THE EYE MUSCLES

DISEASES OF THE ORBIT

1. Diseases of the Eye Muscles

Concomitant Strabismus

Concomitant strabismus is an abnormality of the eyes in which the visual axes do not meet at the desired objective point, i.e., one eye is fixed upon a certain object, while the fellow eye is turned away. The squint may be constantly present in one eye or alternately in both. There is *convergent* concomitant strabismus in which the squinting eye is turned to the nasal side, and *divergent* strabismus in which the squinting eye is turned to the temporal side. A characteristic sign of concomitant strabismus is that the squinting eye has full range of movement. When the normal eye is closed the squinting eye moves to adjust itself to look straight ahead, while the closed eye is turned inwards or outwards according to the type of strabismus.

The most frequent cause of strabismus is refraction anomaly, therefore it should be corrected with spectacles as soon as possible. Children only one or two years old are prescribed spectacles. It should be borne in mind that the squinting eye often has a low visual power (amblyopia) which cannot be improved with spectacles. In such cases it is helpful to exercise the eye, bandaging or patching the fellow eye. Older children with an appreciable improvement of vision in the squinting eye are told to exercise their vision, for example by using a stereoscope for training binocular vision.

Treatment. If therapy fails, and in cases of a large deviation (angle of strabismus), a surgical operation is performed on the muscles of the squinting eye, either to strengthen a weak muscle by shortening it and drawing the site of its attachment closer to the limbus (tenorrhaphy) or to weaken a strong muscle by withdrawing it backwards (recession). The outlook of strabismus is more hopeful if treatment is given early.

Paralytic Strabismus

This form of strabismus is caused by paresis or paralysis of an eye muscle. It is characterized by double vision (diplopia), which is not present in concomitant strabismus, and by complete or partial absence of eyeball movements to the corresponding side. In paralytic strabismus, the eye is also incapable of adjustment. This condition is due to affection of the nuclei of the corresponding motor muscles or their trunks. Special examination procedures are used to find out which muscle is affected.

Treatment is given for the principal disease, and is carried out by a neuropathologist. Sometimes surgery of the eye muscles is required.

Nystagmus

Nystagmus is caused by a spasm of the eye muscles, and is manifested by involuntary oscillatory movements of the eyeball either in the horizontal meridian (*lateral nystagmus*), or in the vertical meridian (*vertical nystagmus*), or else by a partial rolling of the eyeball about the visual axis (*rotatory nystagmus*). Nystagmus may be due to low visual power, congenital or acquired in early childhood, certain diseases of the central nervous system, or affection of the labyrinth of the internal ear.

2. Diseases of the Orbit

The principal sign of an orbital disease is protrusion of the eyeball, which is known as *exophthalmos*. This is commonly accompanied by a sideways displacement of the eyeball and a disturbance in its mobility. These changes are concomitant with an increase in the orbital contents as a result of an orbital tumour, haemorrhages, collection of exudate, oedema of the retrobulbar cellular tissue, thrombosis of the orbital veins, and thyrotoxicosis. Exophthalmos is often due to acute inflammatory processes in the paranasal sinuses or orbital traumas with dislocation of bones and diminution of the orbital cavity. In such diseases exophthalmos is attended by partial blindness due to affection of the optic nerve. Severe dislocation of the eyeball often causes diplopia.

Recession of the eyeball into the orbit, known as *enophthalmos*, is also likely to occur as a result of fractures of the orbital walls with such a dislocation of bone fragments as to cause enlargement of the orbital cavity. Enophthalmos is seen in atrophy of the orbital cellular tissue as well as in paralysis of the sympathetic nerve.

Orbital Periostitis

Orbital periostitis (inflammation of the orbital periosteum) may develop with suppurative processes in the paranasal sinuses, as well as in tuberculosis, syphilis, and after a trauma. The characteristic symptoms are redness of the skin, tenderness of the bony margin, fluctuation and later discharge of pus through a newly-formed fistula.

Treatment is given for the principal disease. Antibiotic or specific tuberculosis or syphilis therapy is given after preliminary penicillin injections. Whenever required, an operation on the paranasal sinuses is performed. The application of heat is helpful.

Orbital Phlegmon

Inflammation of the orbital cellular tissue is attended by exophthalmos, disturbance of eyeball mobility, redness of the eyelids, and fever. The causes of orbital phlegmon are as follows: inflammation of the paranasal sinuses, diseases of the facial skin (erysipelas, furuncle, stye), infected traumas, and infectious diseases. The inflammatory process may extend from the orbit to the cranial cavity, causing meningitis or thrombosis of the brain sinuses which is likely to prove fatal. Orbital phlegmon may end in atrophy of the optic nerve, inflammation and necrosis of the cornea, and purulent inflammation of the inner structures of the eye with ultimate atrophy.

Treatment: sulpha drugs per os, intramuscular penicillin injections, and wide excision of tissues along the orbital margin (orbitotomy) for the drainage of pus. Antibiotics have proved to avert what was frequently a fatal outcome. Vision can also be preserved if treatment is started in good time.

Orbital Tumours

Orbital tumours may be innocent (cysts, angiomata, osteomata) and malignant (sarcoma, carcinoma). The latter, especially sarcoma, may invade the adjoining accessory sinuses of the nose or the cranial cavity. The paranasal sinuses are the favourite site of a tumour, which later tends to invade the orbit.

A characteristic sign of orbital tumours is exophthalmos, sometimes attended by a sideways displacement of the eyeball and disturbance of its mobility. Unlike orbital phlegmon, a tumour gives no inflammatory symptoms or pain and develops more slowly.

Treatment. Surgery is undertaken as early as possible. Benign tumours can be excised with the eyeball left in place, whereas malignant tumours require removal of the entire contents of the orbital cavity (exenteration of the orbit) followed by radiation therapy.

Every case of orbital disease, especially with exophthalmos and eyeball dislocation, requires a detailed examination of the orbital walls and margins, the eyeball and its mobility. Other indispensable procedures are x-ray photography of the orbit, paranasal sinuses and skull, Wassermann's test, and clinical and laboratory examination for tuberculosis. Treatment is given by a specialist.

16

EYE INJURIES

Injuries to the eye and its surrounding parts occur in industry, agriculture, everyday life, and in wartime. These injuries account for 5 to 10 per cent of the total of eye diseases. The frequency of such traumas, their gravity and proportion of the total number of causes of partial or total blindness make this field of ophthalmology very important.

It is necessary to find out in detail the circumstances of injury, i.e., when, on what job and with what object the eye was damaged, whether the injury caused fainting, nosebleed, etc., whether and what medical aid was given. To exclude the possibility of a cranial trauma likely to produce symptoms of a cerebral disorder (vomiting, nausea, loss of consciousness), the patient is examined with a view to his general condition.

Before examining an injured eye, one should carefully remove blood clots from the eyelids, find out if there are haemorrhages, crepitation or damage in the eyelids, or dislocation of the eyeball. This is followed by further examination of the eye after separating the eyelids, using a palpebral levator in the case of a spasm. Inadequate care in opening an eye with a penetrating wound may cause the wound to open, resulting in the escape of the aqueous and vitreous humors and prolapse of the inner structures of the eye. The eyelids should therefore be separated with great caution. Attention is then given to the condition of the conjunctiva, the cornea and the sclera, to the depth and contents of the anterior chamber, the position and shape of the pupil, the crystalline lens, and the ocular fundus. The acuity of vision, the intra-ocular tension (commonly by finger palpation) and the mobility of the eyeball must be determined. If there is the slightest evidence of injury to the orbital walls or a foreign body in the eye or the orbit, radiography of the orbit

and skull is required. All patients with an eye injury are, as a rule, given an injection of antitetanic serum.

Eye injuries may be *mechanical*, *thermal*, or *chemical*.

MECHANICAL INJURIES

Mechanical injuries to the eye may involve the orbit, appendages of the eye and the eyeball itself. Injuries to the orbit and the periorbital parts are variable, ranging from mild traumas of the eyelids with scanty haemorrhage to very severe injuries involving crushing of the eyeball, the walls and contents of the orbital cavity.

For *palpebral haemorrhages*, *subcutaneous emphysema* see Chapter VII.

An eyelid injury with damage to the skin, particularly at the palpebral margin, requires special surgical treatment, i.e., an extremely careful, sparing excision of crushed tissue along the lips of the wound and the application of sutures. These are used to correct the position first of the intermarginal space and then of the lips of the skin wound so that the wound heals without disturbance in the position of the eyelids and their protective functions, entropion or ectropion.

Orbital injuries. Bruising of the orbital region is likely to tear off the trochlea, which transmits the tendon of the superior oblique muscle, and cause diplopia. Injury to the orbital bones may be complicated by displacement of their fragments, which will cause a decrease or increase in the size of the orbital cavity with an associated protrusion or recession of the eyeball. Such a trauma may cause sudden, often hopeless blindness as a result of rupture of the optic nerve, injury to the eyeball with extensive haemorrhage, or ruptures of the inner coats, and is likely to crush the eye altogether. Another factor adding to the gravity of an orbital trauma is the danger of infection causing orbital phlegmon, cavernous sinus thrombosis, and meningitis which threaten the patient's vision and even his life.

Injuries to the eyeball may be *superficial*, *bruising* (*blunt*) or *perforating*.

Superficial injuries are often due to small foreign bodies, such as grains of sand and emery, coal and stone particles, and fragments of metal wedged in the groove of the superior tarsal cartilage or in the superior layers of the cornea. There is a sharp pain in the eye, photophobia and lacrimation. Extraneous particles on the conjunctiva are removed with a piece of wet cotton-wool upon eversion of the eyelid. Foreign bodies in the cornea are extracted with special instruments—a spud or a lance—which are preliminarily sterilized with alcohol or by boiling. Anaesthesia is induced with dicaine.

The eyelids are separated with the forefinger and thumb of the left hand, while the gouge or lance is held in the right hand and brought under the foreign body so as to remove it together with the remnants of rust. This procedure is followed by instillation of an antibiotic solution or application of synthomycin or levomycetin ointment. In isolated cases with marked signs of irritation in the iris, the pupil is dilated (preferably with scopolamine) and kept dilated until the inflammatory symptoms abate (2-4 days). During this period and as long as the pupil remains dilated, the patient should not go to work; the visual power of a dilated pupil is lessened and binocular vision is lost, so that there is a danger of affecting the normal eye. In the case of a deeply wedged-in foreign body which cannot be extracted by the above method, and in complications (corneal ulcer, iritis) following the removal of a foreign body, an aseptic dressing is applied to the eye and the patient is sent to an ophthalmologist.

Superficial injuries not involving the entry of a foreign body may be caused by a tree branch, a straw, a snowball, or a finger, and they usually show a defect in the corneal epithelium—erosion.

Treatment. Administration of antibiotics in solution or ointment form three or four times a day, and application of an aseptic dressing. When there are symptoms of irritation of the eye and a very narrow pupil the pupil should be dilated, provided the intra-ocular tension is normal; special caution should be taken during the administration of atropine to patients above 40.

Healing of the corneal epithelium is favoured by using the patient's own blood in combination with antibiotics. For this purpose, 5 ml of blood is drawn from a vein with a syringe. Several drops are used to irrigate the eye, the remainder being mixed in a bottle with the same quantity of synthomycin or penicillin solution. The patient is told to instill the mixture of separated serum and antibiotics 4 or 5 times a day.

Bruising occurs after the eye has been struck with a stick, a fist, a moving part of machinery, horns of cattle, etc., and frequently causes a serious disturbance. There is a likelihood of haemorrhage in the palpebral tissue, under the conjunctiva, in the anterior chamber, the vitreous body, and the retina. The iris may be stripped off or ruptured and the crystalline lens damaged, with a resulting traumatic cataract or dislocation of the lens (luxation or subluxation into the anterior chamber, the vitreous body and under the conjunctiva).

Signs of lens dislocation are trembling of the iris due to movements of the eyeball and a deepened anterior chamber. In the latter case the crystalline lens looks like a drop of fat.

Bruising may also cause large changes in the internal structures of the eye, such as rupture of the retina with haemorrhage on the

ocular fundus, and rupture and detachment of the retina. Bruising is often followed by secondary glaucoma.

The above changes, especially in the case of extensive haemorrhage and haziness in the vitreous body and oedema, result in a considerable loss of visual acuity. Resolution is likely to be followed by a large recovery of vision provided, however, that no graver lesions are present. Finally, bruising may crush the eye completely.

Treatment involves providing comfort for the eye and applying a binocular bandage. Instillations of atropine or pilocarpine (depending on the value of the intra-ocular tension) are also given. If an injury to the eyeball is suspected an ophthalmologist is required immediately.

Resolution of haemorrhages is favoured by intravenous infusions of sodium iodide or sodium chloride, blood transfusions (in 100 ml doses repeated once in ten days) or autohaemotherapy. Local treatment uses electrophoresis with 5-10 per cent potassium iodide, and dionine by drop instillation or subconjunctivally. If there is damage to the eyeball sulpha drugs are administered per os and antibiotics are given intramuscularly or per os. Whenever required, surgery is undertaken for removal of a dislocated lens, stitching the sclera, excision of a crushed eye, etc.

Penetrating wounds are caused by cutting and stabbing objects, firearms, beaks of birds, explosions, etc. The severity of the damage depends on the size and shape of the injuring object, depth of lodgement of the foreign body, and the site of injury. These wounds may involve the cornea, sclera or limbus.

One of the principal symptoms of a penetrating wound is a decrease in the intra-ocular tension (hypotonia) caused by leakage of the aqueous or vitreous humors. If a wound gapes or the prolapsed inner structures are pressed against its margins, the eye becomes soft like an empty sac. Other frequent signs are haemorrhages under the conjunctiva, in the anterior chamber (hyphaemia) and the vitreous body (haemophthalmos). Corneal and corneo-scleral wounds often involve the crystalline lens. Penetrating wounds may cause loss of vision to the extent of total blindness.

In every penetrating wound of the eye, whatever its history, the orbit and skull should be photographed by x-rays to determine the presence and location of a foreign body. The presence of a foreign body is likely if the iris is ruptured and perforated. In certain cases a foreign body is visible in the anterior chamber, the vitreous body or on the ocular fundus. The prolonged presence of an iron fragment in the tissues of the eye causes pigmentation by a deposit of iron particles (*siderosis*); the iris takes on a rusty colour and rusty spots are seen on the anterior capsule of the crystalline lens. The entry of copper fragments is followed by deposition of copper particles

(*chalcosis*), the crystalline lens showing rainbow-coloured hues and sunflower shaped opacities.

An intra-ocular foreign body causes serious damage to the eye and complicates the course of a trauma. Traumas may be complicated by an infection whose signs are oedema of the eyelids and eyeball conjunctiva, a purulent discharge from the conjunctiva, infiltration of the lips of the wound, and pus in the anterior chamber or the vitreous body. Finally, endophthalmitis or panophthalmitis is also likely.

The most formidable complication of a penetrating trauma of the eye is a sympathetic inflammation of the intact fellow eye, which is liable to occur even after surgery to incise the eyeball and sometimes after diseases involving structural lesions of the eye (for example, perforating ulcer of the cornea). Sympathetic ophthalmia begins at least 10 to 14 days after injury but may also come on within several months and even years of injury. The cause of the disease is so far unknown, and no clear signs exist to indicate the possibility of its occurrence in any particular case. It is known, however, that if a wounded eye fails to heal because of a persistent torpid iridocyclitis, and is hypotonic and tender, the danger of sympathetic ophthalmia is obvious, and the damaged blind eye should be excised.

If a damaged eye has partly retained its visual power, treatment should be continued, and excision considered in relation to the efficacy of treatment and the condition of the normal eye. The disease sets in with a lessening of visual power in the normal eye, deep hyperaemia of the conjunctiva, precipitates on the cornea, opacity of the vitreous body, hyperaemia of the iris, and adhesions at the pupillary margin.

Soon an exudative membrane is deposited in the pupillary region, the pupil is overgrown with scar tissue, and secondary glaucoma appears. Sometimes the only sign of the disease is inflammation of the optic nerve.

Treatment. Patients with severe bruised and penetrating wounds of the eye should be placed in an eye hospital. The earlier the patient is given the required medical aid, the greater the hope of recovery of the eye and vision. Therefore the medical assistant or nurse should instill a penicillin or synthomycin solution, apply an aseptic dressing, inject antitetanic serum and penicillin intramuscularly in a dose of 200,000-300,000 U and send the patient to an ophthalmologist without delay. Should the accident happen in a remote region it is best to deliver the patient in a lying position by plane, after the required emergency aid has been given.

The healing of a penetrating wound is largely favoured by its primary treatment and adjustment of its lips to the best advantage. The ophthalmologist cuts off the prolapsed contents of the eyeball,

which hinder closing of the wound, and sutures it. A magnetic foreign body embedded within the eye is extracted by means of a large electromagnet or a small permanent magnet. X-ray photography is indispensable to locate the site of the foreign body before extraction. In the case of a fresh wound, particularly one involving the crystalline lens, a foreign body can be extracted by bringing the magnet tip close to the wound. A deeply embedded foreign body is extracted, especially when the crystalline lens is transparent, through an incision in the sclera made at a site corresponding to that of the embedded fragment, which is determined by x-rays.

The extraction of non-magnetic foreign bodies is much more difficult and success is less likely.

Therapy of penetrating wounds of the eye involves the instillation of atropine for iridocyclitis or pilocarpine for secondary glaucoma. Local application of antiseptic or antibiotic drugs is indispensable. Other remedies are intramuscular injections of penicillin or streptomycin, tissue therapy, osmotherapy, blood transfusion, x-ray therapy, etc. The same measures are applied for a sympathetic inflammation. Excision of the damaged eye is considered individually in each particular case.

The outcome of a penetrating wound depends on its character, size and location, the presence or absence of infection, and the promptness and adequacy of emergency aid and later treatment. The eye may either heal, retaining many of its functions, or become atrophied. In the latter case it should be excised, especially if signs of sympathetic ophthalmia have been found.

In cases of purulent infection, the ocular contents are removed by evisceration. This operation leaves a stump on which a glass prosthetic eye is fitted two weeks later.

THERMAL AND CHEMICAL BURNS

Thermal burns of the eye and its surrounding parts are caused by the action of fire, molten metal, or hot liquids. Such accidents are common in the home, in industry and in wartime.

Mild burns show nothing but oedema and redness of the skin of the lid or conjunctiva, whereas severe burns cause superficial necrosis of the cornea and conjunctiva (Fig. 78) healing to form adhesions between the eyelid conjunctiva and the eyeball (symblepharon). Opacity of the cornea may be due not only to a burn but also to a disorder in corneal nutrition associated with necrosis of the conjunctiva.

Chemical burns are caused by the action of acids and alkalis. Burns due to acids produce a dry scab which later falls off, leaving

a scar. Acids produce a superficial effect and alkalis a deep cauterizing effect. The sequels of lime burns are especially severe. When sharpening an aniline pencil, particles of slate may get into the eye and cause an eye burn. Aniline readily dissolves in tears and penetrates the conjunctival and corneal tissue, which is likely to cause necrosis followed by coarse adhesions in the conjunctiva. In such cases there is a danger of corneal lesion resulting from direct contact with a chemical and from disturbed corneal nutrition due to a conjunctival burn (Fig. 79).

First aid and treatment. The course and outcome of a burn largely depend on *adequate emergency aid.* First of all, the eyelids are everted so that the palpebral fold may be brought into full view, and all foreign particles detected are removed with a slightly wet cotton tampon. If a burn is caused by aniline particles from an ink pencil, the next procedure is careful irrigation of the conjunctival sac with a 2-3 per cent tannin solution followed by drop instillation of this solution once in every two hours. Should this solution not be at hand for treatment the eye is irrigated with strong tea. In cases of eye burns due to other chemicals thorough irrigation with tap water (1-2 litres) is helpful. For all types of burns this procedure is followed by the application of antiseptic ointments, for example synthomycin ointment, to the conjunctival sac and instillation of sterile cod-liver oil. The instillation of blood in combination with antibiotics is also useful. In extensive necroses of the conjunctiva threatening the formation of adhesions, ointment is applied on a glass stick slipped deep into the fornix to smooth it out and separate the adhesions. In cases of severe burns with extensive areas of dead tissue the patient is given first aid, and then seen

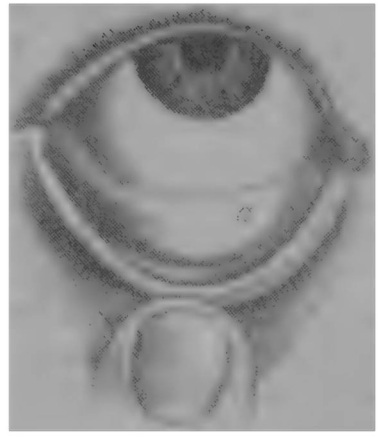

Fig. 78. Necrosis of the conjunctiva after a burn
a—necrosis of the lid conjunctiva

Fig. 79. Burn of the conjunctiva and cornea
a—necrosis of the bulbar conjunctiva

immediately by an ophthalmologist who cuts out dead tissue and grafts a flap of mucosa from the patient's lip to the eye defect.

Atropine is instilled for signs of incipient iritis, provided the intra-ocular tension is normal. It should be borne in mind that burns are apt to be complicated by secondary glaucoma; the intra-ocular tension is therefore watched carefully so that pilocarpine may be administered in good time. If the burns are severe, intravenous infusions of a 40 per cent glucose solution in a daily dose of 10-20 ml are helpful. Vitamins A, B_1, B_2 and C are given per os.

The outcome of an eye burn depends on the severity of damage and the cause of the burn as well as on the adequacy of first aid and subsequent treatment. Some burns heal with full recovery of the conjunctival defect, corneal transparency and visual power, while others leave partial opacities impairing the visual function and are likely to cause a large dense corneal scar (leukoma) with total blindness. Complete destruction of the eyeball, atrophy, adhesions between the lids and between the lids and the eyeball are also likely.

As eye burns are likely to have severe sequels serious attention should be paid to this type of lesion, especially as its gravity is not always seen immediately. Therefore, however mild the signs of an eye burn may look at first sight, vigorous therapy should be started immediately and the patient's condition watched carefully. A patient with signs of a severe burn should be given first aid and seen by an ophthalmologist as soon as possible.

PREVENTION OF EYE INJURIES

An eye trauma requires the most careful attention. A very severe trauma may cause total blindness immediately, while other injuries lead to a large loss of vision making all efforts at therapy futile. A patient with a penetrating wound of the eyeball is threatened by sympathetic ophthalmia, even a long time after injury. If prophylactic measures are to be effective, the causes and character of injuries likely to be sustained on particular jobs should be known exactly. For example, eye injuries in metal workers—turners, fitters, grinders, forgers, etc., as well as in tractor drivers and harvesting combine operators—are caused chiefly by small metal fragments, often burning hot, flying from machined parts and tools. There may be superficial injuries, foreign bodies in the cornea, burns and penetrating wounds, the latter occurring in farm machine operators mostly during repair work. In the coal- and ore-mining industries eye injuries due to fragments of coal and rock are frequent. Damage from bright light is encountered in electric welders and general labourers who neglect eye safety rules.

Many eye injuries are common household accidents occurring during wood chopping, whitewashing, or tending domestic animals.

The following measures are required for the prophylaxis of eye injuries in industry and agriculture:

1. Premises should be cleaned of dust, which is liable to irritate the conjunctiva and cause conjunctivitis so that even a superficial injury may develop into a serious complication.

2. Adequate illumination should be provided in a factory department, particularly in working areas, to relieve visual strain, widen the visual field, aid the colour sense and lessen fatigue.

3. Workers should be instructed in the rudiments of technology to give them a general idea of the design of mechanical tools. They should also be given information on eye injuries likely to follow a breach of safety engineering rules.

4. Regular health education should be given to acquaint the workers with rules for trauma prevention in a particular trade and the required hygienic habits.

5. There should be strict control over the observance of labour protection and safety engineering rules, a rational schedule of working time, prevention of excessive eye strain and visual fatigue at work, attachment of eye shields to machine tools to guard against injuries, and provision of workers with corrigent goggles.

6. A thorough medical examination should be given to applicants for industrial jobs to determine their fitness for a particular trade. Prophylactic examination recording the visual acuity is especially important for workers employed on jobs involving eye strain, because it helps to determine the need for spectacles or a change of trade.

7. Skilled medical assistance should be immediately available in industry and every case of a severe eye injury should be analysed so as to take the required preventative measures.

All such measures consistently carried out in the Soviet Union have drastically reduced the rate of eye injuries in industry. In view of the rapidly growing use of farm machinery, eye injuries likely to occur during the operation of such machinery should also be given adequate attention.

Medical assistants and nurses working at the medical centres of factories, repair shops, and collective and state farms play a leading role in controlling the sanitary standards of factories and plants and the use of individual safeguards, in health education work carried out day after day, and in emergency assistance to accident victims.

Special attention should be given to eye injuries in children playing with sharp, cutting and stabbing objects, such as scissors, knives, needles, slingshots, hand-made pistols, and during explosions of cartridges and mines they have found, etc. Such injuries

are commonly encountered in boys from 6 to 12 years of age. These accidents usually occur in spring and summer when children have more spare time for play.

The prevention of these injuries requires thorough guidance work at school and care of children at home. School teachers and medical personnel should make children aware of the dangers of eye injury. Every case of eye injury should be explained to children and discussed at lessons. In their spare time, particularly during the summer vacation, children should be encouraged to take part in a useful pastime (organized games, excursions, helping their parents, reading). Sharp and cutting objects should be kept where children cannot find them.

Caustics and astringents

1. Rp. Argenti nitrici 0.1-0.2
   Aq. destill. 10.0
   MDS. For cauterization. Not to be dispensed to patients.

2. Rp. Protargoli 0.1-0.4
   Aq. destill. 10.0
   MDS. Eye drops.

3. Rp. Collargoli 0.2-0.3
   Aq. destill. 10.0
   MDS. Eye drops.

4. Rp. Zinci sulfurici 0.025-0.05-0.1-0.2
   Acidi borici 0.2
   Aq. destill. 10.0
   MDS. Eye drops.

Antiseptics

5. Rp. Chinini hydrochlorici 0.1
   sine acido parati
   Aq. destill. 10.0
   MDS. Eye drops.

6. Rp. Sol. Hydrargyri oxycyanati 1.0 : 6,000 200.0
   DS. For eye irrigation.
   Poison!

7. Rp. Sol. Acidi borici 2% 200.0
   DS. For eye irrigation.

8. Rp. Sol. Kalii hypermanganici 1.0 : 5,000 200.0
   DS. For eye irrigation.

9. Rp. Albucidi 3.0
   Aq. destill. 10.0
   MDS. Eye drops.

10. Rp. Ung. albucidi 30% 10.0
DS. Eye drops.

11. Rp. Brilliantgrün 0.1
Spiritus vini 70% 10.0
MDS. For external application.

12. Rp. Ung. Hydrargyri oxydati flavi 1% 5.0
DS. Eye ointment.

13. Rp. Sol. Furacilini 1.0 : 5,000 10.0
DS. Eye drops.

14. Rp. Furacilini 0.02
Lanolini 2.0
Vaselini 8.0
M. f. ung.
DS. Eye ointment

Antibiotics

15. Rp. Syntomycini 0.03
Aq. destill. 10.0
MDS. Eye drops.

16. Rp. Ung. Syntomycini 5% 10.0
DS. Eye ointment.

17. Rp. Ung. Levomycetini 5% 10.0
DS. Eye ointment.

18. Rp. Streptomycini (calcium chloride complex) 100,000 U
Sol. Natrii chlorati 0.85% 10.0
MDS. Eye drops.

19. Rp. Penicillini 100,000 U
Sol. Natrii chlorati 0.85% 10.0
MDS. Eye drops.

20. Rp. Terramycini 0.1
Aq. destill. 10.0
MDS. Eye drops.

21. Rp. Ung. Terramycini 1% 10.0
DS. Eye ointment.

22. Rp. Ung. Tetracyclini 1% 10.0
DS. Eye ointment.

23. Rp. Ung. Dibiomycini 1% 10.0
DS. Eye ointment.

Stimulants and Resolvents

24. Rp. Sol. Dionini 2-4-6-8-10% 10.0
DS. Eye drops.

25. Rp. Kalii jodati 0.1-0.3
Aq. destill. 10.0
MDS. Eye drops.

Anaesthetics

26. Rp. Cocaini hydrochlorici 0.3-0.5
Aq. destill. 10.0
MDS. Eye drops.

27. Rp. Dicaini 0.025-0.05
Aq. destill. 10.0
MDS. Eye drops.

28. Rp. Sol. Novocaini 0.5-1% 10.0
Sterilis!
DS. For intravenous, intramuscular retrobulbar injection.

Mydriatics

29. Rp. Atropini sulfurici 0.1
Aq. destill. 10.0
MDS. Eye drops.

30. Rp. Scopolamini hydrobromici 0.025
Aq. destill. 10.0
MDS. Eye drops.

31. Rp. Homatropini 0.1
Aq. destill. 10.0
MDS. Eye drops.

32. Rp. Plathyphyllini 0.1
Aq. destill. 10.0
MDS. Eye drops.

33. Rp. Phenamini 0.1-0.3
Aq. destill. 10.0
MDS. Eye drops.

34. Rp. Adrenalini hydrochlorici 0.01
Aq. destill. 10.0
MDS. Eye drops.

35. Rp. Sol. Adrenalini 0.1% 10.0
in amp. N. 10
Sterilis!
DS. For subconjunctival injections.

Miotics

36. Rp. Pilocarpini hydrochlorici 0.1
Aq. destill. 10.0
MDS. Eye drops.

37. Rp. Eserini salicylici 0.025
Aq. destill. 10.0
MDS. Eye drops.

38. Rp. Furamoni 0.3-1.0
Aq. destill. 10.0
MDS. Eye drops.

39. Rp. Phosphacoli 1.0 : 5,000 10.0
DS. Eye drops.

Miscellaneous

40. Rp. Cortisoni 0.1
Aq. destill. 10.0
MDS. Eye drops.

41. Rp. Ung. Cortisoni 1% 10.0
DS. Eye ointment.

42. Rp. Riboflavini 0.001
Citrali 0.001
Ac. ascorbinici 0.1
Sol. Glucosae 2% 10.0
MDS. Eye drops.

INDEX

A

Accommodation 39-41
examination of 63
length of 41
mechanism of 40
paresis of 41
power of 41
spasm of 41
Adaptation 35
Adaptometer 35
Anterior chamber 63
Aphakia 138
Aqueous humor 21
examination of 63
Asthenopia accommodative 42
muscular 43
Astigmatism 39

B

Blepharitis meibomian 85
simple 84
ulcerative 84
Blepharospasm 86
Burns 164-166

C

Cataract 135
acquired 136
complicated 139
congenital 135
immature 137
mature 137
overripe 137
primary 136
secondary 138
senile 136
traumatic 138
Choked disc 153
Conjunctiva 15, 18
diphtheria of 100
diseases of 95
examination of 57-61
folliculosis of 168
in trachoma 106-108
Conjunctival sac 15
irrigation of 73
Conjunctivitis 95
acute 96-101
caused by drug allergies 103
chronic 91
diplobacillary 98
due to irritating factors 102
follicular 109
measles 102
pneumococcic 97
Convergence 24
Cornea 17, 18
diseases of 116-128
examination of 61, 116
in trachoma 107
Crystalline lens 21, 132
diseases of 132
dislocation of 139, 161
injury to 161

D

Dacryoadenitis 88
Dacryocystitis 91
in the newborn 94
Dacryocystorhinostomy 91
Diopter 38

E

Electrophoresis 72
Emmetropia 37, 40
Endophthalmitis 120
Enophthalmos 88, 156

Episcleritis 128
Eyeball 11, 12, 17-24
Eye
drops 74
ointments 74
powders 74
Eyelid 13-16
ectropion 86
ntropion 86
erysipelas of 83
examination of 57
inflammatory diseases of 93
massage of 75
oedema of 82, 83
ptosis of 14, 87
subcutaneous emphysema of 82,160
tumours of 88
Exophthalmos 156

F

Fovea centralis 20

G

Glaucoma 140-148
blindness in 144
forms of 140, 141, 142, 146, 148
Gonoblennorhea 99

H

Haemianopsia 34
Haemophthalmos 162
Herpes febrilis 121
zoster 121
Hyperaemia conjunctival 95, 96
pericorneal 96
Hypermetropia 37, 40, 41, 42, 45
Hyphaemia 162

I

Injections subconjunctival 75
Intra-ocular tension 65-67
in glaucoma 145
Iris 18
examination of 63

K

Keratitis 117-122, 125-127

L

Lacrimal apparatus 16, 88
canaliculi 16
caruncle 13
gland 16
lake 13
papillae 16
passages 16, 61, 90
punctum 16, 89, 93
sac 16, 92
Lacrimation 89
Lagophthalmos 14, 87
Leukoma 127, 128

M

Macula lutea 20
Meibomian glands 14
Myopia 37, 42, 43

N

Nyctalopia 32

O

Ocular fundus 63
examination of 64
Ocular media 22
examination of 63
Ophthalmic artery 23
Ophthalmoscopy 64
Optic disc 21
examination of 64
Optic nerve 21, 152
atrophy of 153
inflammation of 153
Ora serrata 19
Orbital fascia 12
fissure 12

P

Palpebral fissure 13
limbus 14
Pannus scrofulous 120
trachomatosus 105, 107, 115
Panophthalmitis 134, 163
Phlyctens 120
Pingueculae 104
Presbyopia 41
Pterygium 104
Pupil 18, 163

R

Refraction 37
Resorption treatment 71
Retina 19, 149, 151, 161

Retinal artery, obstruction of 149
vein, thrombosis of 150
Retinoblastoma 152
Retinopathy in pregnancy 151

S

Schlemm's canal 17
Sclera 17
Scleritis 128
Scotoma 34
Siderosis 162
Skiascopy 44
Spot, blind 20
yellow 20
Spring catarrh 103

T

Tear drainage 16
Tenon's capsule 24
fascia 12
Test, canaliculus 89
collargol 89, 90
Therapy antibiotic 68, 69
derivative 147
desensitizing 70
nonspecific 71
specific 71
sulpha drug 68
tissue 70
ultraviolet light 72
vitamin 69
Thermotherapy 72
Trachoma, complications of 106-108
stages of 105, 106
Transthermia 72
Trichiasis 107, 115

U

Uveal tract 18

V

Vision binocular 35
central 26
colour 30, 31
field of 32
peripheral 32, 33
Visual acuity 27, 30
angle 27
Vitreous humor (body) 21, 22, 63, 139, 162

W

Wall-eye 100, 116, 127, 123

X

Xerosis 107, 115

TO THE READER

Mir Publishers would be grateful for your comments on the content, translation and design of this book. We would also be pleased to receive any other suggestions you may wish to make. Our address is: 2, Pervy Rizhsky Pereulok, Moscow, U.S.S.R.

Printed in the Union of Soviet Socialist Republics